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Contents

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THE RHODE ISLAND MEDICAL JOURNAL

Volume XXIII JUNE, 1940 Number 6

GASTROSCOPY AND CLINICAL MEDICINE

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The gastroscope and the procedure of gastroscopy is beginning to receive universal acclaim. The "miracle" of the gastroscope has been eulogized in both popular and scientific literature. It is perhaps unfortunate that the average physician is no better acquainted with the subject of gastroscopy than the layman who regularly reads his "medicine" at weekly intervals.

Unless the physician is thoroughly familiar with the procedure, or has himself observed the gastroscopic image of the stomach, he is in no position to argue the matter of statistics, the detailed description of mucosal patterns, or the choice of diagnostic nomenclature.

Therefore, the present paper is not concerned with an academic discussion of any specific gastric abnormality or an analysis of cases. Rather it is an attempt to portray by word pictures my impression of gastroscopy, and to tell in general the value and limitations of the procedure.

Visual inspection of the inside of the human stomach tends to quicken the emotions. The observer cannot help but respond to the beauty of the scene — the array of color, the rhythmical contractions of the pylorus, the bubbling pools of secretion, the dancing highlights, and the shifting shadows cast by the rugal folds!

But the very things which delight the artist or stir the poet only too often become a nuisance to the gastroscopist. The pylorus is an impassable barrier; the color may confuse one not versed in art; the lakes of secretion obscure vision; and lurking in the depths of the shadows may rest the lesion he is seeking. For the most part, these handicaps may be overcome by patience, an urge to see, and by skillful manipulation of the instrument.

It is generally conceded that the interpretation of the pictures seen through the gastroscope is the one really difficult feature of gastroscopy. Therefore, it is essential for the observer to become thoroughly familiar with the normal gastric pattern. This may seem an endless task, for even the most experienced gastroscopist will encounter pictures strange to him. The living gastric tissue, as seen through the gastroscope, is in no way similar to the gross specimen removed at operation or at post-mortem.

I will attempt to verbally visualize for you the gastroscopic picture of the normal human stomach. The instrument has been quickly passed down the esophagus and to the distal segment of the stomach. To the onlooker, this may appear as a spectacular feat of skill, but actually it is not difficult. With the ocular sighted, and the light turned on—a blaze of red meets the eye. This means that the mucosa is close to the objective. A small amount of air is now introduced into the stomach. The mucosa is now quickly pushed away and the distal segment comes into view. With the instrument properly orientated an excellent view of the antrum and pylorus may be obtained. The antrum appears as a long, dark tunnel. In a few seconds peristaltic waves may be seen approaching the pylorus and changing the form of the antrum. The contracting waves usually bring the pylorus into view. It appears as a dark, round hole with a smooth edge. Usually, within a few seconds the pylorus begins to open and close. A few bubbles of duodenal secretion may spurt through the open pylorus. The rhythmic working of the pylorus is truly fascinating to behold.

The distended antral segment is practically without rugal folds. The few that are present cast shadows which tend to darken the visual field. The mucosa is of an orange-red color. The observer must feel certain of this, for even slight changes in color may indicate disease. As the ocular is turned from side to side, glistening highlights appear. These indicate a normal moistness of the mucosa.

Read before the Providence Medical Association, February 6, 1939.

From the clinic of gastroenterology, department of medicine, R. I. Hospital.

Drawings by John Parker.

INDICATIONS FOR GASTROSCOPY

1. Persisting gastro-intestinal symptoms. Particularly in those conditions termed "dyspepsia," "bowel distress" or "gastralgia."
2. Unexplained gastric hemorrhage.
3. Persistent anemia associated with gastric distress. Anemia, loss of appetite, loss of weight, (? early cancer).
4. Gastric ulcer. For the differentiation of benign and malignant ulcer. Process of healing.
5. Post-operative study of the stomach. Persistent symptoms may mean — marginal ulcer or gastritis.

When the antrum has been thoroughly inspected, the instrument may be withdrawn a short distance. Now we are looking into the body of the stomach. With proper orientation, the lesser and greater curvatures, and anterior and posterior walls may be inspected. The lesser curvature is without rugae and is the smoothest part of the stomach. The anterior wall is lined with fine, ridge-like folds, while the posterior wall contains a great number of thick, rope-like folds which seem to run in every direction. The greater curvature usually presents a nicely arranged pattern of parallel folds.

Along the most dependent part of the greater curvature may be seen the "mucus lake." The "lake" is an interesting spectacle but also an obstacle as it may obscure vision. The shore of the "lake" is steep and ridge-like; the surface of the pool is often of a bile-like color. Clumps of greyish-white mucus, or even small food particles float about. With a few puffs of air to irritate the stomach and start vigorous contractions, the tranquility of the lake is suddenly disturbed. We now see a rushing, mountainous falls, covered with a snowy foam of air bubbles. The clumps of mucus are helpless and are thrown with volcanic force against the advancing waves. With relaxation of the stomach the lake assumes its usual place and form.

Further withdrawal of the instrument brings into view the dark cavity of the fundus and the cardia. The rugal folds of the fundus are steep and cast shifting, dark shadows. One must proceed with caution for a tiny lesion may be easily obscured by the overlapping rugae. The visible part of the cardia is a pale, almost white, small, rounded ridge lying close to the objective. Due to the rigidity of the walls and the closeness of the mucosa to the objective, the cardia and fundus are difficult to inspect. Additional inflation at this point usually promotes "belching" and restlessness of the patient. When this occurs, it is good judgment to terminate the examination, quickly withdraw the tube, compliment the patient for his show of courage, and instruct him to "belch" at will.

There are certain sections of the stomach which cannot be visualized with the gastroscope. A small area along the lesser curvature of the antrum constitutes the most important of these "blind" spots. This is a distinct misfortune, for this region is frequently the site of important gastric lesions. Lesions at the cardia are apt to be missed with the gastroscope. Any detailed study of this region should be made with the esophagoscope.

It is not difficult for the experienced observer to detect an abnormal gastric pattern. For example, the uniformity of the gastroscopic picture has led to the general acceptance of the term gastritis.

In the past, the term gastritis was loosely employed and did not constitute an exact diagnosis. The symptoms of gastritis are vague, the gastric chemistry variable, and the X-ray findings negligible. The flexible gastroscope affords the one positive means of making an accurate diagnosis.

The appearance of the mucosa will suggest the degree and character of the inflammatory reaction. The process may be one of diffuse superficial gastritis, or progress to a stage of chronic atrophy or chronic hypertrophy. Not infrequently the different types may occur in varying degrees in the same stomach. If the process is chiefly of the superficial type the mucosa will be a dusky or brilliant red color, the folds swollen from oedema, and glary tenacious mucus may adhere to the membrane. The membrane is usually friable and scattered along its surface may be seen superficial erosions. It is quite startling to see drops of blood appear on the surface of an erosion.

If the process is one of hypertrophy, the rugae appear as enlarged, swollen and tortuous folds. The highlights may be absent. Mucus in excessive amounts usually does not occur. The most interesting feature of hypertrophic gastritis is the formation of nodules and nodes. Sometimes these become so large as to be mistaken for true polyps.

The mucosa lying between the folds is frequently heaped up and irregular, looking very much like paving stones. Superficial erosions with active bleeding or superficial ulceration are common.

The gastroscopic picture of atrophy is very characteristic. The normal orange red color of the mucosa is replaced by a grey, greenish grey, or purple. The mucosa is thinned out and the blood vessels of the submucosa become visible. These often spread out and form ramifications with neighboring groups of vessels. The rugal folds are diminished in number and are readily flattened out with air. The atrophic areas are usually "patchy" and much of the mucosa may appear normal.

In clinical medicine gastritis has become an important disease. It is probably the most common disease of the stomach. In our small series of 104 gastroscopies, evidence of gastritis was present in about 60% of the group. The mild, mixed forms occurred most frequently. The patchy, atrophic type was common, while the severe, diffusely atrophic or hypertrophic types were seldom seen. Pigment spots, hemorrhagic streaks, and superficial erosions were frequently encountered. It should be emphasized that gross hemorrhage is a common and important complication of gastritis. There are still many physicians who doubt this. I feel certain that if the skeptic could observe at first hand the fiery red, blood streaked, friable mucosa, or see fresh blood oozing from the surface of an erosion, he would seriously consider gastritis in the differential diagnosis of gastric hemorrhage.

The exact etiology of chronic gastritis is still in doubt. Mechanical or chemical irritants, infection, allergic and neurogenic factors have been mentioned as possible causes. The treatment of gastritis is still inadequate. However, the future of the gastritis problem seems hopeful. Clinicians are beginning to realize that alterations in mucosal structure and physiology are sufficiently commonplace to warrant more than academic interest.

Gastric ulcer is readily detected by the Roentgenologist. However, the common superficial ulcer is an exception. It may be entirely impossible to demonstrate on the screen or film the shallow crater of the superficial ulceration. Such an ulcer may be diagnosed gastroscopically. Furthermore, the exact character of the lesion and the results of medical treatment may be readily determined.

CONTRA-INDICATIONS

1. Obstructing lesion at the cardio-esophageal orifice.
2. Positive disease of the chest — cardiac disease, aneurism of the aorta, angina, dyspnea.
3. Varices of esophagus—large liver.
4. Uncooperative patients. No danger to patient but instrument is friable and likely to be broken.
5. Dysphagia. If the stomach tube encounters persistent resistance—do not pass the gastroscope.
6. A successful gastroscopy requires patience and gentleness. Do not use the procedure when tired or hurried.

Our gastroscopic experience with gastric ulcer is limited, as we have encountered only five instances of chronic gastric ulcer to date. The ulcer is easily recognized as its gastroscopic appearance is characteristic. It stands out as a bright white or yellowish-white spot in an orange red field. It is usually round, the edge sharp and clearly demarcated from the adjacent mucosa, and the floor is covered with a yellowish-white exudate. Occasionally, one may see a slight oedema or redness of the mucosa overlying the rigid walls. The problem is not one of recognition — the real difficulty is one of discovery. This is largely one of location. Ulcers occurring within the "blind areas" will not be seen. Rarely is it possible to visualize a true pyloric ulcer. The dark shadows cast by the large and tortuous rugae of the posterior wall may prevent the discovery of a small ulcer. Fortunately for the gastroscopist, the majority of gastric ulcers occur along the lesser curvature and are readily discovered.

Most ulcers appear as tiny spots. This is a distinct surprise and disappointment to the onlooker, who has expected to see a lesion comparable in size to the niche on the film. The cause of this discrepancy is due to the fact that the divergence of the X-ray beam tends to magnify, while the optical system of the gastroscope diminishes the depth as well as the size of the crater.

TECHNIQUE

1. Fasting.
2. Seconal capsule 1½-3 grs. given 1 hour before examination.
3. Atropine sulphate gr. 1/150. S. C.
4. Codeine sulphate gr. 1/4. S. C. (optional).
5. Pontocaine Sol. 2% as local anesthetic.
6. Use of Hypo-pharyngeal tube optional.
7. Complete removal of stomach contents with Ewald tube.

It has been customary to follow the course of the ulcer with the X-ray. The complete disappearance of the niche indicated healing and ruled out the possible presence of malignant degeneration. Our clinical and X-ray experience has taught us to accept this criteria with some caution. Even a carcinomatous ulcer may show marked diminution in the size of the niche. It is essential that the course of every gastric ulcer be determined at frequent intervals. This entails much time and expense.

On the other hand, the gastroscopist can easily determine whether or not the lesion is benign or malignant. The gastroscopic appearance of cancer is entirely different from that of ulcer. We have observed gastroscopically six instances of gastric cancer. One of the most striking features of the gastroscopic picture of cancer is the vivid coloration of the mass. The multitude of colors is due to the circulation of blood through the tumor mass. The excised specimen is cold and drab as compared to the image of the living mass. The ulcer-cancer presents an irregular base. The color of the wall is usually a dark red, and in sharp contrast to the surrounding pale mucosa. The floor is very irregular and covered with a dirty grey, or brownish-black sloughing exudate. The mucosa adjacent to the ulcer may be "peppered" with tiny greyish-white or reddish nodules. In one instance, the ulcerated area bled so freely as to obscure vision.

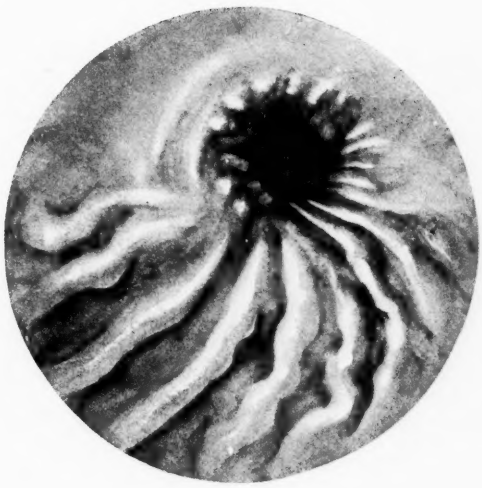
Comment

This brief introductory discussion of clinical gastroscopy has merely "scratched" the surface of an important and highly specialized subject. The clinical value of gastroscopy will of necessity be dependent upon the competence and skill of the gastroscopist. To achieve any degree of competence requires a large experience. To properly evaluate the gastroscopic picture the observer must have carefully studied a great many normal as well as diseased stomachs. The appearance of the normal gastric pattern must be constantly in mind if abnormal changes are to be appreciated. The gastroscopist, above all, must be honest with himself—he must guard against the tendency to see more than is actually within the sphere of vision. Uncertain or imaginary pictures will lead only to improper deductions.

The clinician must carefully study the gastroscopist's report for it may not always be possible, or even feasible, to correlate his findings with the clinical picture. The mechanics of gastroscopy do require a certain degree of skill, but the operator may acquire this through practice. Of even greater importance is sound clinical judgment, for this alone will save the gastroscopist from many a pitfall.

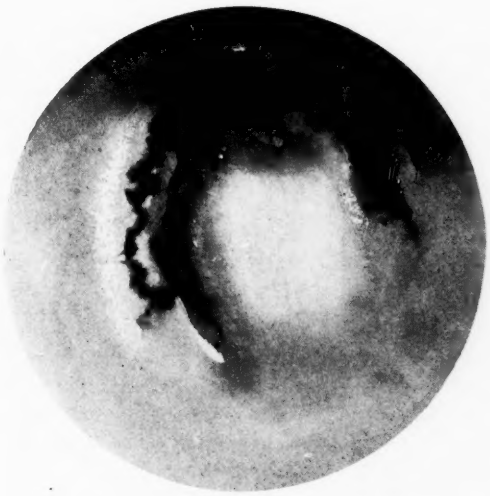
Summary

1. If gastroscopy is to remain a procedure of value, claims for its usefulness must be based on facts. An attitude of reasonable conservatism will be most favorable to the continued progress of gastroscopy.
2. The Wolf-Schindler flexible gastroscope allows the observer to actually see what is going on in the stomach.
3. It is the one positive means of diagnosing gastritis.
4. Gastroscopy allows the differential diagnosis of benign and malignant ulcer.
5. Gastroscopy may reveal the origin of gross hematemesis.
6. It must be understood that the gastroscope will not, and can not, supplant the X-ray in the diagnosis of gastric disease. An experienced Roentgenologist, however, will not hesitate to admit that he frequently encounters mucosal patterns and profile distortions which are of doubtful diagnostic value. Therefore, gastroscopy is always of value in checking X-ray findings. X-ray and gastroscopy are complementary rather than competitive methods of diagnosis.

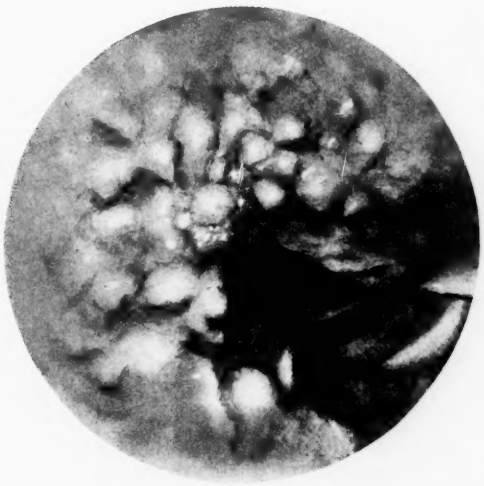


Normal Pylorus.
Process of closing. Highlights.
Rugae formed by peristalsis.

Atrophic Gastritis.
Marked atrophy. Visible blood vessels.
Discolored mucosa.



Gastric Ulcer (lesser curvature)
Benign. Crater seen in distance with shadows.
Hemorrhagic streaks.



Malignant Ulcer (antrum)
Only part of dark floor seen.
Mucosa "peppered" with nodules.

BRAIN ABSCESS WITH BRAIN POTENTIALS*

CHARLES A. MCDONALD, M.D. AND
MILTON KORB, M.D.

PROVIDENCE, RHODE ISLAND

The American method in medical literature is to collect a series of cases and draw conclusions. The Continental way is to study a single case thoroughly. We are reporting in considerable detail a case of brain abscess studied by many physicians, because it was one of the first cases in which brain potentials were used as a helpful clinical laboratory test.

HISTORY OF THE SICKNESS:

B. R., a seventeen year old white American boy, was admitted to the Rhode Island Hospital Neurological Service in September, 1937, with the following history: In February, 1937, he complained of momentary blurring of vision and of feeling the walls of the room coming toward him and going away from him on retiring. It is alleged that three years before, he was knocked unconscious by a blow on the head with a hockey stick. In the middle of July he was hit on the head by a baseball. No investigation was made of these complaints, about which confirmation is not available.

On August 1, 1937, while in a C. C. C. camp, he began to have bursting headaches over the right temple with projectile vomiting and some dizziness, but no nausea. On September 8, 1937, (Ft. Adams Hospital), he was observed as drowsy, moaning and lethargic. On September 11, (Ft. Banks Hospital), blood pressure was 112/68, vision 20/20 O. U., and there was three diopters of choked disk in each eye. A few days later he had a tremor of the right hand which lasted a few seconds.

EXAMINATION OF THE PATIENT:

Rhode Island Hospital Neurological Service (Dr. C. A. McDonald)

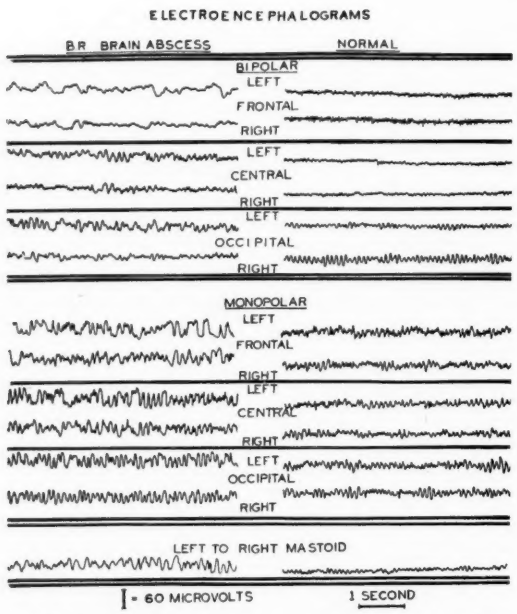
On examination he showed choked disks (four diopters), with retinal hemorrhages, exudate, and some loss of visual acuity. There was bilateral sixth nerve palsy and weakness of the right lower face. Cerebrospinal fluid showed pressure 350 mm. water, no cells, and protein 29 mg. Skull plates were negative for fracture, calcification, and erosion. On October 16, visual acuity was 20/30 O. D. and 20/50 O. S. On October 17, there were bilateral choked disks, double sixth nerve palsy, lateral nystagmus, dysmetria of the left arm, right pupil 4 mm., left pupil 3 mm., bilateral Babinski, Oppenheim, Gordon signs, and right ankle clonus. Kent Intelligence Test showed a Mental Age of 14 years, and a Vocabulary Test (Stanford Binet Word List, 1916 norms) showed a Mental Age of 14 plus.

The diagnosis of "Brain Tumor of Left Cerebrum" was made.

Electroencephalographic examination made on October 17 at the Rhode Island Hospital (Dr. H. H. Jasper and Dr. Korb) gave the following results: (Figure 1) "Electrical activity of the occipital region as determined from both monopolar and bipolar leads appears to be fairly normal, although the alpha rhythm of 8.5/sec. is somewhat low in frequency. This is without significance, however, since we do not know what the frequency was previous to his illness. A few irregularities in rhythm from the monopolar leads to the left occiput obtained as opposed to right occiput. Both bipolar and monopolar leads to the left motor and frontal regions show a definitely pathological condition to exist relative to the potentials obtained from the right motor and frontal regions which were also abnormally large and slow but might be considered at the extreme of the normal. The large amount of potential waves at 7/sec. picked up between the right and left mastoid bones suggests a deep lying pathological condition, especially since this 7/sec. rhythm is also found from the mastoid to motor region leads. In the latter case, however, it is combined with some of the 8.5/sec. cortical rhythm found from bipolar leads which restrict potentials obtained to local cortical areas. No evidence was obtained of strictly localized cortical pathology with the exception of the tendency for abnormalities to be predominately from the left hemisphere and more marked anterior to the central fissure. Pathological waves appear chiefly when recording from the mastoid bone to the top of the head or between the two mastoid bones, suggesting a deep lying pathological condition."

*This report was made possible by the cooperation of the doctors whose names appear in the article.

FIGURE 1



BRAIN WAVES: Left half of figure shows brain potentials from various parts of the head in this patient. Right half of figure shows brain potentials from corresponding regions in a normal individual. Calibrations for amplitude and speed of waves are indicated at bottom of figure. In records of patient note abnormal slow waves, especially from left frontal and left central regions. Such waves are not present in records of normal individuals. For further description see text.

Brain potential records shown in Figure 1 were made in the laboratory of Dr. H. H. Jasper at the Emma Pendleton Bradley Home, East Providence.

Massachusetts General Hospital, Neurological Service (Dr. J. B. Ayer)

On November 9, 1937, he entered the Neurological Service of the Massachusetts General Hospital. Examination at that time revealed: "Cerebrum: clear, well oriented, without memory defect, astereognosis or aphasia. Cerebellum: no nystagmus, adiadochochinesia, or past pointing. Cranial nerves: I—normal. II—marked papilledema, 3 D on right and 4 D on left, visual acuity diminished, more on left. III, IV, VI—internal strabismus on right, internal strabismus and downward inclination on left, no diplopia. V—there is definite weakness of pterygoids on left with deviation of jaw on opening. Sensory V—intact. Motor: left hand and arm seem weaker than normal. No noticeable difference in legs. Sensory: intact, including excellent appreciation of skin writing. Reflexes: increased AJ on left, positive Babinski on right, equivocal Babinski on left. Sympathetic and endocrine: intact."

Electroencephalogram was done on November 11, 1937, (Dr. R. Schwab and Dr. D. Williams): "There are slow waves rising from both cerebral hemispheres. These are greater and slower on the left side and by the use of double cortical leads there appears to be diffused cortical abnormality in the left parietal region, possibly posterior to the fissure of Rolando.

The presence of such widespread slow activity shows obvious widespread cortical impairment. This sort of record can be seen in chronic high intracranial pressures due to tumor, c.s.f. obstruction, or encephalitis. When due to a tumor, the cause is usually deep, and as the abnormality in this case is more marked in the parieto-occipital (left) region, I would surmise that there is a large subcortical neoplasm which is causing unusual damage in this area."

Ventriculogram was done on November 12, 1937, (Dr. J. Michelsen and Dr. J. Keeley): "Bilateral occipital incisions on either side of the midline made. Patient was restless during the procedure and complained of much pain, although we had done the novocaine infiltration as usual. Bone was thick, poor in calcium. Dura opened on both sides and attempts were made to hit the ventricles. I hit the left ventricle without any difficulty right away in its normal position. Fluid came out under high pressure, but the amount of fluid was pretty small. I could not hit the right ventricle at the first attempt, but by putting the needle more to the right I entered the ventricle and fluid came out under high pressure. Apparently the largest amount was on this side. When we tried to fill in with air, there was a strange occurrence of air not passing from the left side to the right, but would pass from the right side to the left. We put in a fairly large amount of air. We did not measure it exactly because the air was evading on the other side into the right ventricle. We tried to fill air into the left ventricle, now the passage was free. Closure as usual with silk. Was this a valve mechanism? The ventricular estimation shows definite evidence that the lesion has to be located above the tentorium. There is also some evidence that it is near the midline and pushing the right ventricular system to the right side. T.P. on fluid from right ventricle 9 mgs. T.P. on fluid from left ventricle 93 mgs."

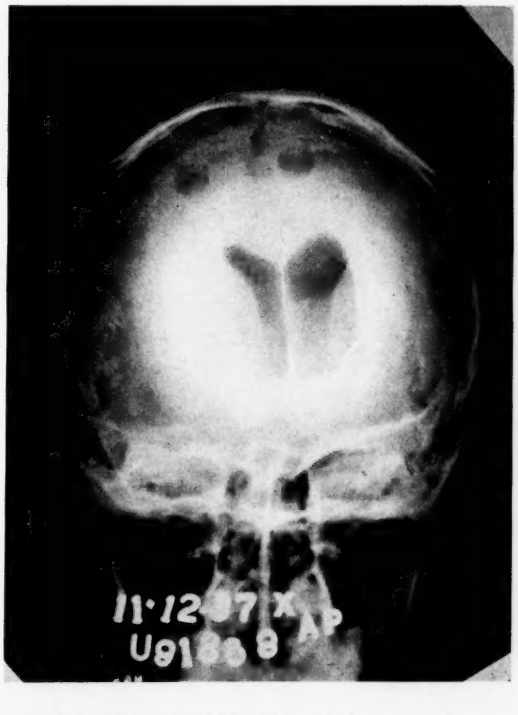
X-ray report on ventriculogram: (Figure 2) "Ventriculogram shows filling of both lateral ventricles and third ventricle. The right lateral ventricle is moderately dilated. There is displacement of the anterior halves of both lateral ventricles to the right. There is amputation of the anterior horn on the left side.

These are consistent with a mass deep in the left frontal lobe, probably fairly close to the lesser wing of the sphenoid."

OPERATION:

Operation was done on November 15, 1937, (Dr. J. Hodgson and Dr. J. Michelsen): "A left fronto-temporoparietal bone flap was laid back by Dr. Michelsen under local anaesthesia. The dura was tense and the ventricle was tapped through the anterior horn, but not much fluid was obtained. No pulsation was felt throughout the dura. On opening the dura there was considerable herniation, flattening of convolutions, and diminished to absent pulsation. At the extreme frontal end of the exposed field the cortex and subcortex appeared yellow and somewhat soft, and on inserting a needle in this region, resistance of an elastic type was met at a depth of about 3 cm. The brain was then opened, and I came on to a discrete, encapsulated lesion. I did not tap it immediately but dissected around it. It appeared to be as large as a plum. As I explored digitally a gush of pus appeared at once, suggesting brain abscess. I then punctured with a fine needle the abscess wall and obtained moderately thick, greenish-yellow purulent material. I made a smear and took a culture. Smear was examined by Dr. Kubik who at first felt that it was more like that of tumor, but finally found a few cells. He did not find any organisms. After a consultation with Dr. Ayer and Dr. Kubik I decided to enucleate the abscess. I was able to get around all of it, except the anterior and lower portion, very easily, but this part was extremely abherent, and it was only after prolonged effort combined with the cutting of a small attachment far anteriorly that I was able to remove the sac about in toto. By this time I had opened into the sac and caused the pus to completely discharge. Bleeding was then controlled and the bone flap was replaced and the wound closed, leaving a Miller wick in the region from which the abscess had been removed. The brain meanwhile had become markedly swollen and closure was difficult. The ventricle was not opened, and at the end it appeared that the chances of spreading infection were not great. Patient's condition was good at the end of operation."

FIGURE 2



VENTRICULOGRAM: Note displacement of anterior halves of both lateral ventricles to right.

Surgical pathology report, November 15, 1937, (Dr. C. Kubik): "Brain: Gross: Brain abscess, inluding the entire capsule, measuring 4.5 x 3.5 x 1 to 1.5 cm. in its collapsed state and 4 x 3.5 x 3 cm. when filled with gauze. The capsule contains a perforation and only a small amount of thick greenish grey pus is found in the cavity. It is composed of dense fibrous tissue and measures from 2 to 3 mm. in thickness. There is little if any brain tissue adhering to it. Microscopic: The outer part of the capsule is dense connective tissue, the inner part granulation tissue. The later contains an unusually large number of large mononuclear phagocytes."

The patient died of meningitis.

AUTOPSY: (Dr. C. Kubik)

"Head. In the left frontoparietal region there are a large suppurating operative wound and a soft fungating mass, the latter protruding through a frontoparietal osteoplastic craniotomy measuring 10 cm. long and 8 cm. wide. The mass, which is partly covered by the elevated bone flap, spreads out in mushroom fashion outside the opening in the skull. Both the outer and inner tables of the skull near the opening are pitted by tiny irregular depressions containing pus.

There is a firm clot approximately 0.5 cm. thick and 3 cm. in diameter, which is dark red and yellowish orange in color, lying between the dura and the inner table of the left frontal bone. It covers the posterior wall of the frontal sinus and the region above it.

Medially and anteriorly in the left orbital plate there is a shallow oblong depression measuring 1.8 x 0.8 cm., with brain and meninges, which are dark yellowish brown in color at this point, bulging down to fill it. Much of the bone at the bottom of the depression is eroded away and a probe passed through the opening in the floor of the depression enters the left frontal sinus by way of a short tract, from 2 to 3 mm. in diameter, a large part of which has a pale, smooth, moist surface as if lined with mucous membrane. The sinus does not contain pus; its lining membrane is greatly thickened and in places raised in rounded elevations.

Extending inward from the anterior medial margin of the depression in the orbital plate to the midline about 3 cm. anterior to the ethmoid bone is a linear fracture in the posterior wall of the frontal sinus. There is no visible separation of the fragments nor any apparent proliferative reaction. The fracture is confined to the left half of the frontal bone and no other fractures are observed.

The right frontal sinus, ethmoid cells and sphenoid sinus are not remarkable.

There is no thrombophlebitis, regional veins, superior longitudinal, cavernous, superior petrosal and lateral sinuses being examined.

Brain. Weighs 1650 grams. In addition to the large suppurating left frontoparietal cerebral hernia and the flat elevation on the left orbital surface there is a thick pale greenish gray subarachnoid exudate which is extremely profuse on the base of the brain and brain stem."

CONCLUSION:

This was a proven case of brain abscess diagnosed as brain tumor. The origin was a skull fracture, not suspected. The case was thoroughly studied. In addition to the neurological examination, mental tests, air ventriculograms and electroencephalograms were done. This was one of the first cases in which electroencephalographic examination was used as a clinical laboratory test.

For the July number of the JOURNAL, Drs. Charles A. McDonald and Milton Korb will contribute a paper on "Brain Tumor with Normal Brain Potentials."

DEDICATION OF OSLER MEMORIAL TO BE HELD AT BLOCKLEY

The old autopsy house where Osler worked at Blockley has been restored as the Osler Memorial Building, and will be dedicated on the grounds of the Philadelphia General Hospital, at Curie Avenue, near 34th and Pine Streets, Philadelphia, Pa., at 2 P. M. on June 8, 1940.

Original furnishings, including the necropsy table, have been collected. The painting by Dean Cornwell, N.A., of New York, entitled "Osler at old Blockley," later to be hung in the building will be on exhibition during the celebration.

There are facilities in the building for the housing and preservation of relics of old Blockley, as well as Osleriana. The Committee would welcome any additions to this collection.

A cordial invitation is extended to those who are interested, and especially those who are planning to attend the American Medical Association Convention in New York City, June 10th to 14th.

CHARLES V. CHAPIN HOSPITAL

On the first of April, Dr. Madeline Burlingame came from the Long Island College Hospital to affiliate here for three months. She is a graduate of Wellesley College and completed Cornell University Medical College in 1938.

Dr. Saul C. Levine of Malden, Massachusetts commenced an internship on May first. He attended Tufts College and received his degree from Tufts College Medical School this year.

POST-OPERATIVE DISTENSION

A SYMPOSIUM

Presented at the Memorial Hospital on Interne Alumni Clinic Day, November 1, 1939

1. CAUSATION OF POST-OPERATIVE DISTENSION
2. DIAGNOSIS OF INTESTINAL OBSTRUCTION
3. TREATMENT OF POST-OPERATIVE DISTENSION

CAUSATION OF POST-OPERATIVE DISTENSION

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At the beginning of the eighteenth century, the classification of abdominal distension was very meagre and text books were wont to describe all pictures of abdominal distension, abdominal pain and associated prostration as Inflammation of the Bowels. As to the cause of this inflammation, quoting George Fordyce, M.D., of the Royal College of Physicians, and Physician to St. Thomas's Hospital in London, in his "Elements of the Practice of Physic," 1771, we find "It is brought on by external cold, indurated faeces, heavy or hard bodies lying in the intestines, introsusceptions, adhesive stimulants, spasmodic contraction of the intestines, hernias and wounds. It takes place also, as other inflammations, in the beginning of fever."

As postmortems were obtained more frequently, as operative technique improved, and more operations were performed, it became apparent that these abdominal distensions could not be classified completely under the old term "Inflammation of the Bowels," for all distentions were not due to primary inflammatory conditions. Ascites, tumors, cysts, hernias, enlarged viscera, mal-formations, enlarged glands, diseases of the genito-urinary tract, pancreatic diseases, a distended bladder, obesity, even normal pregnancy must be considered in the question of abdominal distension. Obstruction of the stomach or intestine is the most important cause of post-operative distension. The term Ileus has been applied to intestinal obstruction from any cause. It was defined by Dorland as severe colic due to intestinal obstruction but in recent years it has come to imply the paralytic form of intestinal obstruction.

Obstruction with distension of the bowel may be due to changes extraneous to the bowel itself; we find abnormalities of the peritoneum causing kinks, adhesions of the peritoneum, hernias, volvulus, growths encroaching upon the bowel wall, diseased omental, mesenteric of retro-peritoneal glands, inflammatory conditions of the peritoneum or of adjacent viscera, abscess formation, and enlargement of other organs causing pressure on the bowel wall.

Distensions may be caused by changes in the intestinal tract itself, such as anomalies of the bowel, diverticulae, intussusception, tumor, tuberculosis, ulcerations and inflammations, obstruction from fecoliths, gall stones or foreign bodies.

Obstruction due to loss of normal function of the bowel from disturbance in its neuro-muscular control is classified as Paralytic Obstruction or Paralytic Ileus. It may arise from trauma, infection, post-operative complications, emboli or other circulatory disturbances.

The intestinal contents are propelled by peristaltic waves of contraction along the muscular coats of the bowel, and at the same time are subject to a mixing process by ring-like contractions which are non-progressive in character.

A stimulus, applied at any point of the small intestine, will produce contraction above the stimulus and relaxation below. These areas of contraction and relaxation travel as waves, 1 to 2 cm. per minute. Since it is proven that peristalsis continues even though the bowel is completely separated from the central nervous system by experimental severance of the splanchnics and para-sympathetic nerves, but that the peristalsis ceases its activity when the walls of the intestines are cocainized, it is thought that the progression of food through the intestinal tract is produced by reflex stimulation

through the nerve plexus of Meissner and Auerbach in the bowel wall itself. The reflex is started from the point of contact between the bolus of food and the nerve endings in the bowel wall and the stimulus carried to the nerve plexus in the wall of the intestinal tract.

The segmenting or mixing action of the bowel is non-progressive, and like the peristaltic wave, continues though it be separated experimentally from central nervous control. Unlike the above, it is not affected by the local use of cocaine, so the impulse does not originate at the point of contact, but probably arises from the distension of the bowel by the bolus of food as it passes along.

The central nervous system exerts a controlling influence upon this independent nerve mechanism of the bowel through the sympathetics and parasympathetics. Stimulation of the para-sympathetic produces contraction of the bowel but with no effect upon the ileo-cecal sphincter. Resection of the vagus has little effect upon the contractions and movements of the intestines. The splanchnic nerves being inhibitory, stimulation results in a cessation of both peristalsis and segmentation, but contracts the ileo-cecal juncture. Division of the splanchnic nerve causes marked increase in intestinal movements due to the loss of inhibitory control. The splanchnics are inhibitory, the vagi are motor. The passage of food into the cecum is thought to be controlled by a gastro-ilial reflex, the stimuli received from the stomach.

Considering that post-operative abdominal distension is generally the result of intestinal distension and that intestinal distension is the result of obstruction to the normal passage of the contents of the bowel, then any of the causes of intestinal obstruction may be the cause for post-operative distension.

Of course, at the time of operation any congenital abnormality is generally seen and if not corrected at the time of the operation, is definitely noted as a possible cause for ensuing complication. Herniations are always possible either into existing peritoneal pockets of congenital nature or through new openings made by the surgeon at the time of operation and left without closure, a definite fault in technique. Separation of the incisional wound in one or more of its layers with resultant herniation may give rise to obstruction and post-operative distension. Here again a small tab of omentum may be left between the edges of the sutured peritoneum with resultant giving away of the sutures terminating in obstructed bowel.

Bands of adhesions resulting from localized peritonitis or foreign body reaction from suture material may either completely or partially obstruct the bowel with resultant distension. The same may occur from bands and adhesions between omentum, peritoneum or bowel itself and broken down glands, generally of tubercular origin.

Peritonitis may cause distension from mechanical obstruction through the formation of adhesions and bands, as may abscess formations.

Pressure from enlarged organs such as kidney, liver or spleen may be responsible for distension by direct pressure. If exploration is possible at the time of operation, these enlargements may be determined. I have fairly recently seen a post-operative distension arising from a rapidly enlarging spleen due to thrombosis of the splenic vessels. I have also seen distension arising from volvulus about the anastomosis of a posterior-gastro-enterostomy. That this might happen shortly after operation is certainly possible.

Regarding the pathology of the intestinal tract itself, the anomolies, diverticulae, tumors, tuberculosis, inflammations, foreign bodies, fecoliths, may generally be seen at the time of operation. Intussusception may occur post-operatively as demonstrated by a case operated upon for intussusception, the intussusception reduced, and a second intussusception occurring post-operatively at an entirely different portion of the bowel with complete cessation of symptoms between the two calamities.

Trauma plays an important part in the production of the paralytic form of obstruction, and may arise from injury to the spine, ribs or abdomen. It is thought the distension is reflex, from the abdominal wall peripheral nerves to the central ganglia, stimulating the splanchnics or inhibitory nerves, the derived inhibition overcoming the normal intestinal activity. Thus we reason that not only mauling of the abdominal contents but lack of care in the use of retractors and unnecessary handling of the abdominal wall itself may result in paralytic ileus. I feel that this is most important.

Peritonitis from contamination of the peritoneal cavity or other causes, may result not only in obstruction from bands of adhesions, sharp angulations, or gluing together of loops of intestines, but the paralytic type of obstruction may be produced through the chemical toxic effect of the peritonitis upon the peripheral nerves with production of a reflex to the sympathetic splanchnic nerves with cessation of peristalsis and segmentation. The stimulation may also be directly upon the splanchnic nerves, or upon the bowel wall itself, or through the blood stream. For the same reasons we find paralytic ileus occurring at times in the course of some of the infectious diseases, pneumonia, typhoid, sepsis, meningitis. It is thought, also that a long narcosis may be instrumental in the production of the ileus through a toxic effect upon the central nervous system.

Blockage of the blood supply to the intestinal loops by thrombosis or embolism may result in post-operative distension through the disturbance in the blood supply with possible gangrene of the bowel. The distal colon receives its nerve supply from two sources: (1) The inferior mesenteris plexus, probably sympathetic in character and (2) from the hypogastric nerves and plexuses which probably carry both sympathetic and para-sympathetic elements but with the para-sympathetic influence predominating. The colon may be involved in paralytic ileus, but the results of the obstruction of the colon alone, unless the cause be from rare vovulus, herniation with strangulation or intussusception confined to the colon, as far as symptoms go, are not of immediate concern. It is not often encountered as post-operative distension, unless it be where a closed loop first stage colostomy has been performed for low obstruction and decompression of the bowel has purposely been delayed. Hirschsprungs Disease is thought to be congenital in origin and possibly under nerve control. Some of the excellent results from sympathectomy support this theory. Fecal impaction may cause post-operative distension which may not be recognized until the dire results of perforation of the bowel with peritonitis have been reached.

The nerve supply of the stomach is from the three sources, the sympathetics, the para-sympathetics and an intrinsic nerve supply. Similarly to the intestinal tract, marked dilatation of the stomach may occur following injury or trauma from abdominal operations. Its early recognition and treatment are essential. Much of the gas found in the stomach and intestinal tract post-operatively is thought to be the result of swallowing of air.

From this discussion it is apparent that most of the causes of intestinal obstruction with its resultant distension may be the causation of post-operative distension. The experimental work on paralytic ileus and the sympathetic and para-sympathetic control of the intestinal tract is far from complete. The relationship of the adrenals is not known. Though great strides have been made in the past ten to fifteen years in the study of intestinal obstruction, regarding water balance, blood chlorides, alkalosis, exchange of gaseous contents of the bowel, toxemia and the cause of death, there are at present many conflicting statements and much left unsaid.

DIAGNOSIS OF INTESTINAL OBSTRUCTION

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The second phase of this discussion of distension implies the consideration of obstruction. This has to do with the morbid anatomy and physiology of obstruction. A. J. Cokkinis has devised the following classification of Acute Obstruction:—

SIMPLE OBSTRUCTION

(A) *High Small Gut*

1. Acute Dilatation
2. Congenital Pylor. Stenosis
3. Spasm
4. Foreign
5. Jejunal obstruction (P. O.)

(B) *Low Small Gut*

1. Adhesions
2. Stricture
3. Gall Stones
4. Foreign Bodies
5. Spasm
6. Paralytic Ileus

(C) *Large Gut*

1. Carcinoma
2. Diverticulitis
3. Fecal Impaction
4. Stricture
5. Congenital Atresia
6. Spasm
7. Hirschsprung's Disease

STRANGULATION

By Bands	Orifices
(A) *Internal* (5)	
1. Peritoneal	Internal Hernia
2. Omental	Richter's Hernia
3. Visceral	Holes in Omenta
Volvulus	Infarction
1. Small Gut	Embolism
2. Cecum	Thrombosis
3. Colon	Trauma
Intussusception	
Primary	
Secondary	

(B) External

1. Inguinal
2. Femoral
3. Umbilical
4. Others

I want to point out particularly the main divisions. First we have all obstructions divided into two chief classes:

1. Simple obstruction classified according to its anatomical site: High Small Gut
Low Small Gut and
Large Gut

2. Obstruction by strangulation, which in turn is divided into

Internal, with five various types, and

External, comprising those arising at the common hernial sites, and all others of less frequent occurrence.

In simple obstruction, the outstanding change is distension above the block. It is progressive. Its degree and rate of increase, vary with the level, duration, and cause. It is most marked in low small gut and large gut type. We have all seen the cecum blown up to the size of a toy balloon with walls of paper-like thinness, friable, and tearing readily. It is at first pale, then congested and cyanotic, due to compression of the vessels from accumulation of fluid and gas. In the later stages there are areas of hemorrhage and there may be actual gangrene at the antimesenteric border of the distended loop in its terminal portion. Peritonitis can occur with or without perforation at these points. The intestine below the block is emptied by strong peristalsis early in the obstruction. The striking difference between the dilated gut proximal to the obstruction and the bowel distal to it always serves as a guide to the lesion. In paralytic ileus, this is less marked.

In the case of obstruction from strangulation, however, the gut above and below the obstruction, while showing essentially the same pathological changes as in simple obstruction, tends to be less distended proximal to the lesion. This is due to the shorter duration of life in the strangulated type, as for instance in strangulated hernia. The distension is rapid due to the inability of the vessels to absorb CO_2 and other gases arising from decomposition of the contents. The strangulated coil becomes congested and cyanosed. Hemorrhagic infarction appears, and finally gangrene. There is usually associated bleeding into the peritoneum as well as into the lumen of the gut. These changes, of course, are more rapid in the internal types.

It is interesting to note here, that Richter's Hernia, which is one of the internal types of strangulation obstruction, involves only a portion of the circumference of the bowel lumen. The constriction is marked and gangrene or perforation may occur early. More important is the fact that there may be few if any obstructive symptoms.

Outside the intestine a fluid exudate accumulates in the peritoneal cavity. In simple obstruction this is serus but with internal strangulation producing the obstruction it soon becomes bloody. When peritonitis develops, it is purulent. The lungs in fatal cases show a broncho-pneumonia. Occasionally gangrene or abscess of the lung may be found. Dehydration changes in the tissues occur. Toxemia, while not prominent in simple obstruction, produces changes in some cases of strangulation which at least suggest the possibility of the toxin theory as a factor in the too often fatal outcome. Neither the theory of bacterial toxemia nor anatomical grounds alone can wholly account for the early collapse and fatal results of acute obstruction.

The physiological changes are variable and many. The level and type of obstruction exert a marked influence on the morbid physiology, even to the point of overlapping between levels and types.

Proceeding then to a consideration of these changes under the main types as shown in the classification of Cokkinis we have: *First:* simple obstruction high in the small intestine. This is the picture of profuse vomiting, severe shock and early collapse suggesting a physiologic disturbance of great gravity. The fundamental changes responsible for this, as recent experimental research has proved, are three: (1) Dehydration of the blood and tissues; (2) prolonged loss of inorganic electrolytes from the plasma, as well as other chemical and physical blood changes; (3) Failure of renal function.

The dehydration is reflected by the intense thirst, dry skin, sunken eyes, hoarse voice and oliguria which occur in high obstruction. This extreme water loss follows not only from the vomiting but also from the complete failure of intestinal absorption due to the high level of block. It was felt at one time that saline infusions exerted their chief beneficial action in the maintenance of water content of the blood and tissues. In the light of later acquired knowledge it was realized that other substances beside water were lost to the body. Blood chlorides in particular suffer in high obstructions as well as other ions such as sodium and bicarbonate. Experimental investigation has shown beyond doubt that the fall of essential plasma salts can only be explained by the loss of digestive secretions from the stomach, pancreas, liver and intestines. It was further proven that the fixed base, mainly sodium, chlorine, bicarbonate and potassium, in the combined digestive juices, exists in proportions closely approximating the ionic content of the plasma. Normally five litres of digestive secretions pour daily into the stomach and upper gut. This is about twice the total volume of the blood plasma which is the source of supply. It is easy therefore, to see the necessity for reabsorption of these juices by the bowel in order that life may continue. Their loss by vomiting, together with the absence of absorption, represents depleted plasma. This loss with associated dehydration provides the explanation of morbidity in high obstruction. With plasma depletion the blood shows concentration and increased viscosity. There is a rise in the red count. The total volume of plasma may be diminished by one-third, yet this big loss represents only a fraction of the total withdrawal of fluid and salts from the body. In later stages alkalosis appears. The plasma protein rises from a normal 7% to as high as 10 or 11%, while the blood urea and non protein nitrogen show increased values. This anhydremia then interferes with the respiratory and renal exchanges of the blood. The impending renal failure with its diminishing urinary output and its lowered excretion of urea and salts may progress to complete anuria with its consequent uremic manifestations.

Second: The morbid physiology of low small gut obstruction as compared to high small gut obstruction appears in the clinical course. When the lesion is low the patient's condition may remain more or less stationary for a number of days and then sudden shock and collapse appear to terminate life abruptly. Two physiological changes are suggested in low gut obstruction, distension and sudden decompression. Proximal distension is the outstanding pathological change in low small gut obstructions and is produced by an accumulation of fluids brought down from above by peristalsis plus the local production of fluid and gases. There is increased secretion of intestinal juices and mucous in the congested and obstructed loop. The tension thus produced compresses the capillaries of the intestinal wall hindering reabsorption and thus setting up a vicious circle. Sudden release of this distension is a real danger and a rather gradual decompression should be attempted for the reason that death often follows a too rapid emptying of the distended gut. These deaths have been ascribed to the overwhelming toxemia, the toxins being absorbed by the healthy intestine beyond the obstruction or the peritoneum. The release of tension opens up compressed capillaries and veins in the bowel wall which through lack of tone become engorged with a resulting loss of blood to the general circulation. Too, sudden decompression may operate through the nervous system to produce mesenteric shock as a contributing cause of death.

Third: Acute obstruction of the large bowel in more than 90% of cases occurs as a gradual culmination of a chronic obstructing lesion. The acute stages are never as urgent as in small bowel pathology partly because the block is of an intermittent,

temporary nature and rarely complete, and partly because the colon has had time to adapt itself to the developing statis. Dehydration and blood changes are never prominent and although extreme distension may occur the large intestine tolerates it better than the small. The danger of sudden decompression is present to a much less degree for obvious reasons.

So much for the simple obstructions. When strangulation is a factor the changes are much more urgent. The crux of this situation is the rapidity with which strangulation causes death. The outstanding features are necrosis, perforation and peritonitis, with accompanying pathological physiology already mentioned operating with greater rapidity and intensity.

The fate of a patient with acute intestinal obstruction depends on an early and accurate diagnosis. Accurate clinical diagnosis involves 4 steps:

1. The discovery of the presence of obstruction,
2. Recognition of the type (simple or strangulated),
3. The level of obstruction,
4. The determination of the cause of the obstruction.

The symptoms of obstruction usually are: (1) Pain; (2) vomiting of a persistent nature; (3) caprostasis or bowel block. Inspection may or may not reveal distension, but this must not be waited for because it is in only large gut obstruction and paralytic ileus that abdominal distension is of real diagnostic value. More significant and earlier are the signs of distension in individual coils. The phenomenon of visible peristalsis when accompanied by pain is to all practical purposes diagnostic. Auscultation of the abdomen is helpful in locating the lesion for certainly the contrast between the turbulance of a mechanical obstruction and the dead silence of paralytic ileus or peritonitis often times is marked. A tumor mass is of value in the search for cause rather than in the diagnosis of obstruction. Rectal examination should never be omitted, for apart from the discovery of local growths, one may feel the pelvic viscera or perhaps find distended loops or tumors in the lower ileum or pelvic colon. And finally X-ray is of great aid in localizing the level of an obstructing lesion.

TREATMENT OF POST-OPERATIVE DISTENSION

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Drs. Davis and Shaw have given us very excellent papers dealing with the etiology and differential diagnosis of post-operative distension. I shall endeavor to outline the treatment of this condition. A few days ago I picked up a pamphlet in our staff room giving in some detail, results obtained by the use of a popular drug for intestinal stimulation. As I read this through, I thought what a Surgeon's Paradise if all of these things could really happen most of the time. To give you some idea of the present diversity of opinion relative to the treatment of post-operative distension, Babcock, in an article on Prevention and Management of Post-operative Intestinal Incompetence writes, "Peristaltic Stimulants" are dangerous as a rule. "Continued distension with evidence of obstruction usually should be treated by one or more enterostomies in proximal distended loops of bowel. Enterostomy has received ill-deserved condemnation from its failure to relieve advanced forms of septic peritonitis or ileus associated with blood stream infections." Engel, in an article on The Treatment of Post-operative Adynamic Ileus, writes, "enterostomy, in form of jejunostomy, we never use, as this will drain only a single segment of the bowel and we have abandoned it."

In reviewing the treatment of post-operative distension, I will discuss first: Treatment of the distension as such, and second: Treatment of body pathology arising from distension. I will not discuss here the prophylactic treatment of distension as I take it for granted we are dealing with the condition already existant. Next consideration, then, would be a study of intestinal stimulants most commonly used in distension. Physostigmine or eserine has been used for many years with varying results measured by the enthusiasm or distrust of the individual user. According to findings of Ochsner, Gage, and Cutting, an active stimulation of the motor mechanism of the intestines at laparatomy in experimental animals was noted. Most investigators feel this is particularly true in the small bowel. It also, however, acts as a cardiac and respiratory depressant. Prostigmin, a synthetic preparation, is now commonly used in place of physostigmine, as it is claimed to be less toxic.

Prostigmin is supplied in 1 cc. ampules of 1-2000 and 1-4000 solution. The 1-4000 solution is recommended for actual treatment of distension and 1-2000 for prophylactic treatment, one ampule every four to six hours for four doses. Harger and Wilky at the Cook County Hospital, Chicago, report excellent results in a series of 175 cases using a 1-4000 solution every two hours for six doses with low incidence of by-effects.

Pituitrin and its derivatives have been used with much favor as intestinal stimulants, but recent investigation by McIntosh and Owings have noted slight relaxation or no change at all following pituitary injections in both normal and obstructed loops of bowel. Peristaltine, a glucoside of cascara sagrada, is another familiar drug used for intestinal distension. Ochsner, Gage, and Cutting find it of little or no value experimentally.

Morphine, in earlier days, was used to relieve pain but believed by many observers to be the cause of intestinal paralysis. More recent experimental study by Plant and Miller and others have shown that morphine increases the tone and peristalsis particularly of the small intestine.

My own observation with intestinal stimulants has been most disappointing, with the exception of morphine which is indispensable. I repeat, morphine alleviates pain and increases peristalsis. One important point to remember is that it constricts sphinctor muscle. Consequently, a small rectal nozzle should be kept in place for escape of gas.

The most effective procedure in the treatment of distension and vomiting is generally conceded to be decompression by tubal drainage. Westerman in 1909 was the first man to use a duodenal tube to relieve post-operative distension. This was followed by Levin in 1921 with a small tipped duodenal tube for nasal intubation. About ten years later, Wangensteen first reported successful decompression of three cases of mechanical bowel obstruction by means of suction drainage. In 1938, Ravdin first suggested using the Miller-Abbott double lumen tube in distension and intestinal obstruction.

The Levine tube is familiar to most of us, being from 45-60 inches long and varying in caliber from 18-26 french with four or more eyes at its distal end. This tube is passed into the stomach readily and, with some difficulty in experienced hands, through the duodenum into the jejunum. Wangensteen added suction drainage to the Levine tube by creating a vacuum or negative pressure in allowing water to flow from a higher container bottle to a lower bottle tapping in a third container and the Levine tube into the vacuum by air tight fittings. This method of drainage is of the greatest value in distension and obstruction. Swallowed air is now believed to play an important role in distension of the stomach and intestines. This important phase is at once overcome by tubal drainage. After the stomach and duodenum have been thoroughly emptied, the Levine tube may be clamped off for twenty minutes immediately following fluid by mouth. This allows passage of liquids through the duodenum and into the small intestine as 90% of fluids are absorbed in the jejunum and ileum and not in the stomach. Clamping of the tube may give distress in the early hours of drainage and if so, immediate suction must be restored. A careful record of all mouth intake and all tubal drainage plus any vomitus will give most valuable information. It may be asked why vomitus if we have suction drainage, but this can and does occur in severe cases. This intake and output plus urinary output is most important in the evaluation of water balance, to be discussed subsequently.

The Miller-Abbott tube is a double lumen rubber tube 16 french in diameter. A rubber septum extends through its entire length. One side is used for inflation and the other lumen for suction. The inflation tube opens into a soft rubber balloon. The suction tube has several openings at its distal end and terminates in a metal tip. As this tube passes the pylorus the balloon is inflated and peristalsis carries it down the entire length of small intestine, unless of course, it meets an actual mechanical obstruction. As it passes along, suction is applied to remove fluid and gas from each distended loop. X-rays will determine the probable position of the tube and assist in localizing any absolute obstruction. We have not yet used the Miller-Abbott tube at the Memorial Hospital but definite results are beginning to appear in the literature.

We now come to the value of enemata in post-operative distension. Surely, most of us have used everything from soap and water to milk and molasses. Peculiarly enough as we are returning to morphine for an intestinal stimulant, many observers are recommending milk and molasses rather than the more irritating soap suds and peppermint

concoctions for enemata. The value of enemata in reducing distension is now believed to have been very much overestimated. An enema, to be sure, will assist in emptying the lower colon of gas and fecal material, but post-operative distension is as a rule in the stomach and small intestine. I feel very certain up to the present time that many of us have exhausted sick and toxic patients with repeated enemata to no avail.

The use of oxygen for gas distension would seem to have a place in theory at least. It is very hard to measure the actual benefits of this procedure. Fine has shown that high concentrations of oxygen (95%) can be given over long periods without harm and should be used in conjunction with the duodenal tube. Reducing the percentage of nitrogen in the inspired air decreases the nitrogen tension in the blood plasma. The nitrogen in the bowel content is then more readily diffused into the venous blood system and exhaled.

Surgical intervention in the treatment of post-operative distension is an ever impending possibility but one in which the exact time is very hard to determine. Dr. Frank Lahey has said that it is safe to wait four days where there is a non-mechanical distension and obstruction before surgical intervention. No hard and fast rule can be made as each case is dependent upon the symptoms presented and the condition of patient.

Simple enterostomy, if indicated, is the most favored procedure and as a rule the simpler the better. Occasionally it is possible to locate a portion of collapsed bowel and free an adhesion. Usually much more damage is done by overexploring than by the so-called "blind enterostomy." Any entero-anastomosis is dangerous in an acutely inflamed and distended intestine. In all enterostomies the loop of distended ileum should be brought outside the abdominal wall. The abdominal incision is closed as well as possible without constricting the bowel. A small opening is then made and a catheter sutured in place for constant drainage. This gives immediate relief of intramural pressure and drainage of toxic material. There are many who will claim this drainage affects only that particular loop of bowel incised, but in spite of this contention the results are sometimes most gratifying. Of course, this should be in conjunction with our other aids, particularly duodenal suction decompression. It is well to irrigate through the catheter with warm normal saline every two hours as this will stimulate bowel tone, peristaltic action, and help to remove any toxic material that is banked in the loop. Any retention of saline in the loop after irrigation may be absorbed to assist in the upkeep of body chlorides. Different absolute distensions and obstructions must be treated individually following any improvement after enterostomy. Cecostomy and appendicostomy have a definite place in relieving mechanical distension of the large bowel. A report of three cases will illustrate the successful use of ileostomy in treatment of post-operative distension. The first case of paralytic ileus caused by adhesive peritonitis not relieved by Wangensteen drainage:

Mrs. I. S., age 41, operated for rectocele, umbilical hernia and chronic appendicitis. No difficulty experienced during operation which lasted one hour and a half under gas-ether anesthesia. *First day post-operative*—complained of gas pains and was moderately distended. Vomited twice in small amounts. *Second post-operative day*—vomited 10 ounces and still distended. Morphia was given gr. 1/6 at four hour intervals. Two ampules of prostigmin were given and the Levine tube inserted for drainage. Removed at end of nine hours because of discomfort. 800 cc. of 5% glucose in saline was given twice daily intravenously. Daily enemata were given with some gas and fecal result. *Third post-operative day*—was less distended. Vomited twice total of 10 ounces, one ampule of petressin was then used at 4-6 hour intervals. During the night vomiting and distension increased. Wangensteen drainage was then made constant but distension continued. Finally on the eighth post-operative day with temperature 100, a pulse 120 and respiration 25, a citrate transfusion was completed. A right rectus incision was made using gas oxygen ether anesthesia. Loops of distended ileum were seen very much adherent with a greyish exudate, no free pus seen. One loop brought outside abdomen. Wound closed. Intestine opened with escape of gas and a greyish watery fluid. Catheter sutured in opening. Patient's condition was very poor that night and most of the next day. Twenty-four hours after enterostomy distension decreased. Good drainage was established through catheter. Patient continued to full recovery with a resection of the ileostomy three weeks later.

Case II E. C. age 15, Toxic paralytic ileus following gangrenous 1 drain appendix. Patient was in constant pain and increasing distension in spite of suction drainage, morphia and intestinal stimulants. On the fourth day post-operative with a pulse climbing from a base of 100 to a base of 130, and a ballooned shiny abdomen, a left rectus incision was made with ½% novocaine supplemented by gas-oxygen anesthesia. The small intestine was very much dilated and injected, but loops were not adherent to each other as in previous case. A loop was brought through abdominal wall. Abdomen closed as much as possible. A suction trochar introduced and a catheter sutured in place with considerable escape of gas and some fecal mucus fluid. The following day pulse came down to 120, distension was markedly less and there was free drainage through enterostomy. This was followed by complete recovery with a resection of ileostomy 3½ weeks later.

Case III E. W. age 39, a mechanical ileus following operation for perforated duodenal ulcer with three drains. Patient improved for nine days post-operatively when he began to vomit and became very much distended, in spite of duodenal drainage and intestinal stimulants. On the 14th post-operative day with a pulse of 135 and temperature of 101 was operated through median left rectus incision. The ileum was distended and a hard mass in the pelvis found, involving several loops of the small intestine under which pus was suctioned. It was impossible to free the loops and an ileostomy was done on a loop of proximal distended gut. This patient fully recovered. As the pelvic abscess drained, a normal intestinal flow was established. The catheter in the ileum drained freely and fell out on fifth day. Eventually the opening in ileum closed and the loop later replaced into the peritoneal cavity.

In the care of the bodily requirements associated with distension, we have to consider three essentials, food - water - chlorides. With our improved methods of suction drainage superimposed upon a non functioning gastro intestinal tract, the water balance of the body is quickly and essentially damaged.

Food requirements may be easily and adequately taken care of with intravenous administration of either 5-or 10% glucose.

Water requirements are great because of loss of fluid by vomiting, perspiration and kidney function. Coller sets the requirements of water at between 2500-3500 cc. per day. The exact measurement of intake and output in these patients is important.

Chloride loss is important since associated with the above fluid loss is chloride loss. Chlorides can also be administered through the intravenous route. Blood chemistry determination will inform us as to the degree of chloride needs. It is important to keep the chlorides to between 450-550 mgm per 100 cc. of blood. The rule to follow is 0.5 gram of salt for every 100 mgm less than normal, per kilo. of body weight. In the debilitated patient in whom it is desired to increase the oxygen carrying capacity of the blood, transfusion is of value. In the intravenous administration of any of the above mentioned fluids, the rate of flow is of importance. A rate of 50-60 drops per minute is felt to be safe.

Summary

The care of the patient suffering with distension is along two major lines. First, care of the distension itself, and second, care of the disturbed physiology associated with distension.

In the care of the former—the following methods have been considered—the use of the intestinal stimulants, physostigmine, prostigmin, pituitrin, and peristaltine. The value of morphine as an intestinal stimulant has been mentioned.

The use of 95% oxygen, and intestinal drainage by the Wangensteen, and the Miller-Abbott tubes have been considered. In furthering the idea of intestinal drainage, ileostomy has been discussed and three case studies presented.

The patient's bodily requirements as dextrose, water, and chlorides have been reviewed. The indication for transfusion has been presented.

REFERENCE

BABCOCK: Surgical Clinics of North America, 1938.

CUTTING: Principles of preoperative and postoperative Treatment.

WANGANSTEEN: Intestinal Obstruction, American Medical Journal, 1933 & 1938.

WISE, LEWIS, & AXELMAN: American Journal of Surgery, 1936.

WISE: Miller-Abbott Tube In Intestinal Obstruction; American Journal of Surgery, 1938.

WANGANSTEEN: Acute Bowel Obstruction; N. E. Journal of Medicine, 1938.

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THE RHODE ISLAND MEDICAL JOURNAL
Medical Library Building
106 Francis Street, Providence, R. I.

ANNUAL MEETING OF THE STATE SOCIETY

To the physicians of Rhode Island comes this month the privilege of attending the annual meeting of the State Medical Society in Providence on June 5th and 6th, and the convention of the American Medical Association in nearby New York the following week.

Attendance at medical meetings becomes habitual to most alert physicans. The varied programs of district and state societies offer the general practitioner a refreshing opportunity to keep posted on new and useful developments in the many clinical fields with which he must be constantly familiar. These same varied programs serve to keep the specialist up to date and informed in medical areas beyond the limits of his own restricted practice. The wealth of clinical information, general and specialized, which is spread before all doctors attending the annual meeting of the American Medical Association would be dazzling and confusing were it not so efficiently arranged for the convenience of the visitor.

Many physicians think of medical meetings entirely in terms of formal papers and addresses by medical leaders. However the personal and informal contacts enhanced by clinics, demonstrations and exhibits at all the larger meetings constitute a major attraction to many practitioners. Here is provided an opportunity to develop intimate professional contacts which are later self-perpetuating. These are especially valuable to the research worker and the administrator to whom a personal exchange of ideas and suggestions is of prime importance.

Attendance at all professional gatherings entails some sacrifice of time lost from practice by a busy doctor. Conventions held at distant points involve the additional expense of time and money for travelling. By many successful medical men such expenditures are considered as investments rather than sacrifices, the direct yield being increased knowledge and experience, and the eventual profits more successful practice and advanced professional prestige. The fact that the most eminent and busiest medical leaders continue to organize and attend professional gatherings in the face of mounting responsibilities of administrative work, research, and clinic practice speaks for itself.

At the June meeting of the Rhode Island Medical Society an invigorating program of scientific papers at the Medical Library will be amplified by clinics and demonstrations at a number of hospitals in and about Providence. An expanded and more attractive array of commercial exhibits will enable the visitor conveniently and comfortably to become directly acquainted with many recently developed drugs and devices. A large attendance is expected and it is hoped that no Rhode Island physician will overlook this excellent opportunity to widen his span of professional vision and participate in reunion with his colleagues.

CANCER EDUCATION

A short while ago the newspapers published President Roosevelt's proclamation designating April as Cancer Control month. This proclamation has a special significance in that it shows on the part of the public a profound and increasing interest in cancer. For the President acted not at the request of the Medical Profession but rather at the request of a rapidly growing group of lay individuals who believe that much can be done to lessen mortality

from cancer by educating the public along cancer lines. This group has as its name the Women's Field Army. It is not an independent organization; it is a part of the American Society for the Control of Cancer. The parent society was formed in 1913 and for years tried to carry its message of cancer education to the public by Field Workers, by so-called cancer weeks held in various parts of the country, and by the distribution of literature. Its success was not striking. Its contacts were too superficial. It had no means of really getting hold of people. Then some bright mind thought of getting the women interested and immediately the situation changed. The Women's Field Army was formed. In state after state energetic women have taken hold as "Commanders," have organized the cities and countries under captains. Each group has a talk on cancer by some medical man at least once a year and during April an intensive drive is carried on.

Membership in the Army costs one dollar. Thirty cents of each dollar goes to the National Society to pay administration costs. Seventy cents stay in each state to be used in whatever way the Executive Committee thinks best.

This Executive Committee is made up in part by the Cancer Committee of the state Medical Society, so that whatever is done is under medical supervision and control.

The Managing Director of the National Society is Dr. C. C. Little, a man known throughout the world for his work in cancer research. The Rhode Island Medical Society is fortunate to have him on its program for the June meeting. We trust he may tell us in considerable detail the progress of the work in cancer education all over the country and the results we may expect in lowering cancer mortality.

THE PRESIDENT'S HOSPITAL PLAN

No one can doubt that a great need for increased hospital facilities exists in many parts of the United States. An attempt to remedy this lack, along with an improvement in medical care of the people generally was launched on a grand scale by the introduction of the Wagner Bill (S-1620). This bill has been widely criticised and it is evident that it will not be passed. The need, however, for which it was designed still exists.

President Roosevelt has recently suggested as a preliminary measure the expenditure of $10,000,000 for the construction of hospitals in communities where they are needed. This has been introduced in the Senate by Senator Wagner (S-3230) and may be considered as an introductory experimental effort of the federal government on a modest scale. Public hearings have been held before a senate committee and representatives of the medical profession have stated their views. It is evident that the bill meets with general approbation.

The plan envisages the establishment of hospitals in communities where need can be demonstrated and is desired and not elsewhere. It is of interest to Rhode Island to note that among the excerpts from letters received from State Health officers pertaining to this bill (and published with the reports of the hearing in the Journal of the A. M. A. April 6, 1940) is one from Dr. Lester J. Round stating, "There is need for 250 beds in tuberculosis sanatoriums. No general hospitals are required." In this state as in many others existing hospital facilities are adequate and are capable of considerable extension if necessary. The erection of a federally owned institution under such circumstances would be distinctly detrimental. Under the terms of the act, however, these hospitals can only be established where they are needed, desired and will be supported.

Following this an amendment was introduced on April 18 by Senator Taft. This proposes certain definite and valuable improvements. In particular it involves an annual appropriation of $10,000,000 for which from 40 to 90 per cent of the cost of construction of new and improvement of existing hospitals shall be provided. It involves also expenditures for the maintenance of these hospitals and that the title shall lodge in the state rather than the federal government. It increases the importance of the National Hospital Advisory Council.

The provisions of this amendment have been based on the suggestions which were received at the hearings on the original Wagner-George bill (S-3230) from the representatives of medical and hospital organizations including the American Medical Association. In the form given it by this Taft amendment the President's hospital plan represents a most timely and valuable piece of legislation and should receive hearty support.

RHODE ISLAND FRACTURE COMMITTEE

Realizing the need for improvement in the treatment of fractures throughout the United States, the American College of Surgeons has formulated a program which is functioning in all but two of the states.

About four years ago Regional Fracture Committees were appointed, corresponding to the geographical division of the country, and the Rhode Island Committee is a component part of the New England Regional Committee on Fractures and Trauma. Each state chairman was asked to appoint members to serve for his state, and these appointments were ratified by the American College of Surgeons. In Rhode Island each general hospital was chosen as a centre for developing and improving fracture treatment in that community. Accordingly one or more representatives were appointed from the staffs of these hospitals and the Committee was constituted as follows: Rhode Island Hospital, Murray S. Danforth and Peter Pineo Chase; St. Joseph's Hospital, William A. Horan; Homeopathic Hospital, Henry McCusker; Miriam Hospital, Simon G. Lenzner; Memorial Hospital, Roland Hammond and Herbert E. Harris; Notre Dame Hospital, William A. Horan; Woonsocket Hospital, Augustine W. Eddy; South County Hospital, John Paul Jones; Westerly Hospital, John W. Helfrich; Newport Hospital, William A. Stoops.

This Committee has promoted the program of the American College of Surgeons by requesting that each hospital maintain: 1) a fracture service, 2) a chief for fractures, or 3) a fracture consulting committee. The governing bodies of the various hospitals have cooperated in this project, and all three programs are operating in Rhode Island hospitals, governed by local conditions in each community. Fracture equipment has been improved and more or less standardized. Ambulances and State Police vehicles are fitted with proper arm and leg emergency splints and first aid dressings, and instruction in the emergency splinting of fractures has been given to ambulance drivers, hospital orderlies, and in some communities to policemen and firemen. Red Cross classes in First Aid instruction have provided many trained volunteer workers and this organization has established First Aid Stations at strategic points. The veterans' organizations have cooperated by providing ambulance service in some communities. Emphasis is placed on the danger of improper handling of an accident victim. "Don't move an injured person until skilled aid arrives" is advice particularly pertinent in fracture cases. "Summon an ambulance immediately and then wait until it arrives — or until a surgeon or someone else competent to render first aid appears on the scene" is the instruction given in First Aid courses.

The Rhode Island Committee on Fractures feels that the time has now arrived for further publicity and education of the medical profession and the lay public in the emergency treatment of fractures in this state. At a recent meeting of the Committee it was voted that the sponsorship of the Rhode Island Medical Society for this program should be obtained. The subject was presented to the House of Delegates of the Rhode Island Medical Society at its meeting on May 16, 1940, and the following endorsement was obtained:

> VOTED: "That the House of Delegates of the Rhode Island Medical Society approve the program of the Fracture Committee of the American College of Surgeons for the emergency treatment of fractures in cooperation with the Red Cross, the Rhode Island Division of State Police, the Division of Motor Vehicles, the Governor's Safety Council, the Department of Education and other related organizations."

A pamphlet describing first aid treatment in fractures of the head, the upper extremity and the lower extremity is now being prepared by a special committee and will be published for distribution to organizations concerned with the public safety.

The Committee plans to act in an advisory capacity to these agencies in order that the emergency treatment of fractures in this state may be placed on a high plane of service to the community.

It is our firm opinion that the program of any lay organization seeking to institute the emergency treatment of fractures should obtain the endorsement and cooperation of the Rhode Island Committee on Fractures, and the Committee stands ready to volunteer its services as a body or individually to give advice or render aid in promoting this undertaking, looking to improved fracture treatment in this vicinity.

ROLAND HAMMOND, M.D.
Chairman, Rhode Island Regional Committee on Fractures, American College of Surgeons.

STATE INFIRMARY

The State Infirmary at Howard, Rhode Island, is now in the hands of an able, efficient Superintendent, Dr. R. P. Crank. For many years it was in the doldrums, shunning publicity, dispensing alms to hapless people who had no homes, no future, no folks to shelter their declining years. The stigma of spending one's last days at Howard in the State Infirmary was a real dread to many an oldtimer in Rhode Island. All this has been changed; care of the poor who are too sick to work and have no relatives able or willing to support them, has ceased to be its chief function. New buildings, newly organized staff, opportunities for constructive medical care have transfused new life into the State Infirmary.

Good surgery, good obstetrics, good medical care, able consultants, assure the Infirmary's patients of very adequate treatment. Patients are kept here longer than at a general hospital, to insure complete recovery, for the people who leave here must go back to work as wage earners or housewives. New paint on the walls and tasteful interior decorations are factors in building a new atmosphere of hope for people who are very near to the giving up of all hope. There are private rooms for acutely ill patients; the operating rooms sparkle with the latest equipment in instruments, lights, and operating tables. Dr. Crank has gathered together a staff of men freshly graduated from their hospital internships, representing many of the leading medical schools in the country.

Physicians may send emergency cases here and within one hour or less, depending upon the location of the patient, they are receiving skilful treatment, either surgical or medical as the case may demand.

Patients who are not willing to accept charity may pay for their hospital care through their local director of public aid. This cost is about one third of the cost of ordinary ward care at any of the general hospitals.

Why not change the name of the "State Infirmary," perhaps perpetuating the memory of one of Rhode Island's public spirited citizens and thus in one act remove the onus of the term "State Infirmary?" Rhode Island has a fine hospital for the care of the sick, state supported and run by well trained efficient physicians. We should use it for the purpose for which it is intended.

PAWTUCKET MEDICAL ASSOCIATION

April Meeting

The regular monthly meeting of the Pawtucket Medical Association was held in the Nurses' Auditorium of the Memorial Hospital, April 18, 1940.

The meeting was called to order by the President, Dr. G. Raymond Fox, at 9:05 P. M.

A communication was read from the Providence Medical Association stating that Drs. F. A. Webster and B. S. McKendall were transferred to associate members and were now eligible for active membership in the Pawtucket Association.

A communication was read concerning the act for reorganizing the Social Welfare Department now pending in the Legislature. The secretary was instructed to express the approval of the Pawtucket Medical Association of the act with changes suggested to the Executive Secretary of the Providence Medical Association.

A communication was read from the National Physicians Committee for the extension of medical services asking for support of this organization. No collective action was taken.

Dr. Henry reported for the committee on compulsory membership in the State Society. A motion was made and seconded that the President appoint a committee to send letters to the individual members to determine their attitude towards this measure.

Dr. Eugene Field was then introduced by the President. Dr. Field gave a very interesting and instructive talk on "The Practical Implications of Serum Protein."

Meeting adjourned at 11:15 P. M.

Collation was served.

Respectfully submitted,

JOHN H. GORDON, M.D.,
Secretary.

PROVIDENCE MEDICAL ASSOCIATION

April Meeting

A regular meeting of the Providence Medical Association was held at the Medical Library on Monday, April 1, 1940.

In the absence of both the President and Vice President, Dr. Harry C. Messinger served as President pro tem for the meeting.

In the absence of the Secretary, the Executive Secretary read the minutes of the last meeting, and on the motion by Dr. Jesse Mowry they were accepted.

Dr. Messinger announced that the President of the Association had received an invitation for the members of the Providence Medical Association to attend a lecture to be given by Dr. Kazanjian of Boston at the quarterly meeting of the Rhode Island State Dental Society to be held at the Peters House on Tuesday, April 30. The invitation to the meeting was extended by Dr. Raymond L. Webster, President of the Rhode Island State Dental Society.

The Executive Secretary reported for the Executive Committee as follows:

It was moved that a resolution endorsing the annual April cancer campaign be prepared and presented to the membership at the regular meeting on April 1.

A resolution proposed by the Committee on Tuberculosis was read and accepted for presentation to the membership at the next regular meeting.

Dr. Peter Chase presented the final draft of an insignia drawn by Mrs. Howard Day after long study and preparation. It was moved and accepted that the insignia be officially adopted for the Providence Medical Association, and that a vote of thanks and appreciation be extended to Mrs. Day for her splendid work for the Association.

Dr. J. Merrill Gibson, Chairman of the Committee on Legislation, presented a detailed report of the work of the Legislative Committee of this Association which has co-operated with the State Medical Society in studying and acting upon legislation before the Rhode Island General Assembly. On a motion from the floor, the report was accepted and placed on file.

The Executive Secretary reported that the Executive Committee had recommended to active membership Dr. Robert A. Clark. Dr. Jesse Mowry moved the election of Dr. Clark and the motion was seconded and unanimously passed.

Dr. T. W. Grzebien presented the following resolution with a motion for its adoption:

Whereas much publicity has been given in the local press to high cancer mortality rate in Rhode Island, and

Whereas the Providence Medical Association has always been eager and willing to lend its full support to any campaign for the elimination of disease from the community.

Therefore, this Association, in meeting assembled this first day of April, 1940, does hereby fully endorse and support the educational campaign conducted throughout the year, and in particular during the month of April, by the American Society for the Control of Cancer, and the Women's Field Army of Rhode Island.

This resolution was unanimously adopted and placed on file.

Dr. Philip Batchelder, Chairman of the Committee on Tuberculosis presented the following resolution:

Whereas the death rate from Tuberculosis in this Community still remains high, and

Whereas the Providence Medical Association has continually pledged itself to the work of controlling this disease, and

Whereas the Providence Tuberculosis League has stated in its annual report that the most important contribution to the work of Tuberculosis control in the community during 1939 was that of the large number of private physicians practicing in the district who referred individuals to the League.

Therefore, the Providence Medical Association, in meeting assembled this first day of April, 1940, pledges its continued enthusiastic co-operation and support to all agencies in the State engaged in combatting Tuberculosis, and further, fully endorses the annual April public educational program of the State and National Tuberculosis Associations.

On a motion from the floor, this resolution was unanimously adopted.

The business part of the meeting being completed, Dr. Messinger introduced Dr. Burton E. Hamilton, Cardiologist at the Boston Lying-In Hospital, who spoke on the topic, "Heart Disease in Pregnancy." Following the presentation of this interesting paper, discussion was initiated by Dr. Frederick C. Irving, Wm. L. Richardson, Professor of Obstetrics at Harvard Medical School and Visiting Obstetrician at the Boston Lying-In Hospital. The following members of the Association also participated in the discussion: Drs. Henry L. C. Weyler, Frank T. Fulton, and Bertram H. Buxton.

The meeting was adjourned at 10:30 P. M. Collation was served.

Respectfully submitted,

JOHN E. FARRELL,
Executive Secretary,
In the absence of the Secretary.

TENTATIVE PLANS FOR A NEW ENGLAND ACADEMY OF MEDICINE

The urgent need of better medical postgraduate facilities has been recognized for several years. Numerous studies have been made by governmental agencies as well as by both professional and lay organizations. Everybody agrees that postgraduate education should be more universal. The medical profession is better qualified and equipped than any other group to take the initiative and organize this much needed activity. An adequate postgraduate medical educational program in this era calls for complete mobilization of all available teaching facilities, materials and personnel if the profession proposes to meet its public-health responsibilities and fully serve the modern civilized community. The mass intellectual level and community demand for better medical service will hardly tolerate a less comprehensive project.

With the above in mind the following project entitled "The New England Academy of Medicine" is submitted for consideration, to be sponsored by the state medical societies and related organizations of the New England states.

PURPOSE: To foster interest in and provide for continued postgraduate education among the medical profession for all time by whatever means is best suited to meet individual and group needs.

ACTIVE INDIVIDUAL MEMBERSHIP: Active members of each state medical society would automatically be active members of the New England Academy of Medicine.

ASSOCIATE INDIVIDUAL MEMBERSHIP: Membership to be open to all legally registered physicians who are not members of organized medical societies; such members would be associated without voting privileges.

INSTITUTIONAL MEMBERSHIP: Active institutional membership would include medical schools, medical libraries, teaching hospitals, special medical societies such as the New England Surgical Society, the New England Heart Association, the New England Pediatric Society and all similar specialty groups which are organized with an avowed active educational purpose as part of their activities.

ASSOCIATE INSTITUTIONAL MEMBERSHIP: This would include non-teaching hospitals that might wish to have their facilities used for teaching now or at a future time. State dental and nursing associations and similar related groups might be considered eligible.

HONORARY MEMBERSHIP: Such to be elected by board of governors.

FUNCTIONS: First, to foster a larger and continuing interest in all present useful postgraduate educational facilities. Second, to improve newly started activities such as the extension courses and the New England Postgraduate Assembly. Third, to aid medical schools in creating larger interest in any postgraduate facilities they may offer and possibly suggest new ones. Fourth, to promote publication and use of all possible forms of inexpensive and easily read medical articles, as well as the more intricate and costly types of medical literature such as would be found in medical libraries; also, aid in wider use of more graphic educational methods. Fifth, co-operation with state and federal agencies interested in postgraduate education of physicians, and in all matters of public health.

ORGANIZATION: The formal organization could be composed of a rather large board of regents representing all the groups, and a smaller board of governors or trustees appointed by the organized medical societies and active institutional members, the former an advisory body and the latter an official body, the whole to be incorporated as a non-profit educational institution. The trustees could appoint an executive committee to actually carry out the policies and program of the organization.

This form of organization would preserve the integrity of old established institutions and in no way interfere with their usual functions; at the same time it would allow all such individuals and organizations to enter new fields and effect a higher level of usefulness by planned and active co-operation.

LOCATION: A central executive headquarters in Boston with operating units in each state and in such smaller units of each state as is deemed best after proper study of field conditions. If and when desirable, such an organization might very well be housed in a building where medical, dental, nursing

and associated groups could be under one roof and have easy access to and acknowledge of their closely related activities.

FINANCES: No study has been made of ways and means of financing such an organization. It seems much more important to conceive adequately the functions and ways of going forward. It is reasonable to believe that once a practical scheme is evolved it can be mobilized by proper underwriting. The reorganization and expansion of the New York Academy of Medicine were so effected just prior to 1925 when it moved into its new and enlarged quarters.

BOOK REVIEW

CHEMOTHERAPY AND SERUM THERAPY OF PNEUMONIA. By Frederick T. Lord, M.D., Elliott S. Robinson, M.D., Ph.D., and Roderick Heffron, M.D. pp. 174. Cloth $1.00. The Commonwealth Fund, 41 East 57th Street, New York City, 1940.

This book is the third in a series of handbooks on pneumonia published by the Commonwealth Fund. The first appeared in 1936 under the title Lobar Pneumonia and Serum Therapy, and the second in 1938 under the title Pneumonia and Serum Therapy. The present book includes a discussion of chemotherapy as well as information relating to the combined use of sulfapyradine and antiserum.

The book is well written, exceptionally meaty, and replete with numerous supporting references. The authors collected data showing that sulfapyridine has given brilliant results in all types of pneumonia, in early and in late cases. They present convincing evidence that in certain cases judicious use of serum and drug therapy may be expected to lower death rates still further. A plea for more thorough laboratory checks is made in the chapter on diagnosis. Pneumococci account for 96% of pneumonia cases, and 60-70% of these are due to types 1, 2, and 3. Most frequent types in their order were: 1, 3, 2, 5, 8, 7, 4, 14, 9, 18 and in infants and children 14, 1, 6, 19, 5, 4, 3, 7. Death rates varied directly with day after onset that treatment is begun, and the number of lobes involved. Infancy, childhood, old age, and winter were significant factors. Precautions through proper history of drug and serum sensitivity, sensitivity tests, and adequate laboratory work to properly guide efforts in drug or serum therapy, and as a means of indicating when the one or the other or both are necessary is stressed. This book contains many practical hints and should be extremely helpful to every man who treats a case of pneumonia.

SCOTT L. TARPLEE, M.D.

RHODE ISLAND MEDICAL SOCIETY

Program for the One Hundred and Twenty-Ninth Annual Meeting

June 5–6, 1940

Morning Sessions

Wednesday, June 5

Homeopathic Hospital of Rhode Island
Chalkstone Avenue, Providence

8:30 In Operating Room

Eye, Ear, Nose and Throat Surgery
WILLIAM M. MUNCY
FRANK MACCARDELL

Anesthesia
JOHN HAYWARD

9:30 General Surgery

1. Radical Breast Amputation following Pre-operative Radiation

Post-Operative Demonstration

1. Carcinoma of Descending Colon
2. Melanoma of Leg

ROBERT H. WHITMARSH
JOHN HUBBARD

Anesthesia
RICHARD ALLEN

10:00 Urology *Out-Patient Department*

Clinical Demonstration of Bladder Closure after One Stage Prostatectomy
EDMUND A. SAYER
JACK SAVRAN

10:30 Dermatology *Out-Patient Department*

1. Psoriasis
2. Alopecia Areata
3. Allergic Dermatitis

HARALAMBIE CICMA

11:00 Bone Tumor Symposium *Out-Patient Department*

1. Multiple Myeloma
2. Osteogenic Sarcoma involving tibia
3. Metastatic Lympho-sarcoma. Pt. lived 7 years. Spontaneous healing of bone lesions
4. Lymphoma of Hodgkins type in Sternum
5. Extra-Osseous Osteogenic Sarcoma in Muscle

DRS. MCCUSKER, MCKENDRY, SCHRADIECK AND HUNT

Providence Lying-In Hospital
Maude Street, Providence

9:00 to 10:00 Inspection of Hospital

10:00 to 12:30 Dry Clinic

10:00 to 10:20 Review of last 12 years at Providence Lying-In Hospital
Manual Removal of Placenta
CHARLES POTTER

10:20 to 10:40 Trend in Caesarian Sections
ALFRED L. POTTER

10:40 to 11:00 Preliminary Report. Follow-up Clinic of Toxemia of Pregnancy
JOHN G. WALSH
WALTER JONES
ROBERT MURPHY

11:00 to 11:20 Review and Discussion of the Use of Bags for the last 12 years in the Providence Lying-In Hospital
CRAIG S. HOUSTON

11:20 to 11:40 Report of 3 Cases of Congenital Atresia of Esophagus
MAURICE ADELMAN

11:40 to 12:00 Manikin Demonstration of Operative Obstetrics
GEORGE W. WATERMAN

Morning Session
Thursday, June 6

Butler Hospital
Blackstone Boulevard, Providence

SYMPOSIUM ON INSULIN AND METRAZOL THERAPY

9:45 Recent Progress
DOUGLAS D. BOND
(Discussion to follow)

11:00 Technique and Complications
WALTER C. WEIGNER
(Motion picture and X-ray demonstration)

11:30 Clinical Demonstrations
IRA C. NICHOLS
(Observation of patients in Treatment)

Memorial Hospital
Prospect Street, Pawtucket
Round Table Discussions

SURGICAL SERVICE:

Under the direction of Frederic V. Hussey *Chief of the Surgical Division*

10:30 to 12:30

1. Biliary Tract Disease
FREDERIC V. HUSSEY
The following men have been invited to attend: William P. Davis, Lucius C. Kingman, Frank E. McEvoy, Emery M. Porter, Wilfred Pickles, Robert H. Whitmarsh, Robert Williams, R. S. Bray, E. W. Benjamin, James F. Boyd, Elihu Saklad, Louis I. Kramer, Frank B. Cutts, George L. Young, John C. Ham, Stanly D. Davies, Albert H. Miller.

10:30 to 12:30

2. Peptic Ulcer
ELIOT A. SHAW, *Senior Surgeon*
The following men have been invited to attend: William A. Mahoney, Charles J. Ashworth, Charles O. Cooke, Robert R. Baldridge, Henry B. Moor, B. Earl Clarke, Eugene A. Field, Isaac Gerber, Lawrence A. Martineau, John A. Hayward, Francis H. Chafee, Robert G. Murphy, Paul C. Cook, Irving A. Beck, Albert A. Barrows, Meyer Saklad.

OBSTETRICAL SERVICE:

10:30 to 12:30

Prolonged Labor
JOHN G. WALSH, *Chief of the Obstetrical Division*
To be discussed by: Edward S. Brackett, Bertram H. Buxton, Alfred L. Potter, George W. Waterman, Herbert C. Partridge.

UROLOGICAL SERVICE:

10:30 to 12:30

EYE SERVICE:

10:30 to 12:30

Medical Ophthalmology
RAYMOND F. HACKING, *Chief of the Eye Division*
To be discussed by: George W. Van Benschoten, Joseph L. Dowling, Frank J. McCabe, Harry C. Messinger.

PEDIATRIC SERVICE:

10:30 to 12:30

Asthma in Children
EARL F. KELLEY, *Chief of the Pediatric Division*
To be discussed by: William P. Buffum, Henry E. Utter, Banice Feinberg, Reuben C. Bates, Lucy E. Bourn, Francis V. Corrigan.

SKIN SERVICE:

Under the direction of William B. Cohen and Vincent J. Ryan

10:30 to 11:30

1. Treatment of Acne Vulgaris
WILLIAM B. COHEN

11:30 to 12:30

2. Treatment of Occupational Dermatosis in Relationship to Compensation
VINCENT J. RYAN
To be discussed by: Carl D. Sawyer, F. Ronchese, Roswell Wilcox, Malcolm Winkler.

MEDICAL SERVICE:

10:30 to 12:30

Under the direction of John F. Kenney, *Chief of the Medical Division*

1. Treatment of Pneumonia with Sulfapyridine
JACOB GREENSTEIN, *Visiting Physician*
To be discussed by: Alex M. Burgess, William S. Streker, Halsey DeWolf, Charles F. Gormley.

10:30 to 12:30

2. Treatment of Edema
RAYMOND E. STEVENS, *Junior Assistant Physician*
To be discussed by: Frank T. Fulton, Guy W. Wells, Elihu S. Wing, Herman A. Lawson.

NOSE AND THROAT SERVICE:

Under the direction of Francis B. Sargent, *Chief of the Nose and Throat Division*

10:30 to 11:30

1. Management of Sinusitis
N. A. BOLOTOW, *Assistant Surgeon*
To be discussed by: Linley C. Happ, Gordon J. McCurdy, Francis L. Burns.

11:30 to 12:30

2. Meningitis of Otitic Origin
FRANCIS B. SARGENT, *Chief of the Nose and Throat Division.*
To be discussed by: Howard E. Blanchard and Frank M. Adams.

TUMOR CLINIC:

10:30 to 12:30

Under the direction of John F. Kenney, G. Raymond Fox, Jesse P. Eddy, 3rd, and Associates.

ORTHOPEDIC SERVICE:
10:30 to 12:30

1. Demonstration by the Fire and Police Department of Pawtucket and Central Falls of First Aid for Fractured Spines and Fractured Legs to show application of Thomas Splint.
2. Exhibition of Photographs of Types of Fractures.
3. Moving Picture of the Method of Reducing Colles Fracture and Treatment.
4. Round Table Conference on Fracture of the Spine

Afternoon Session
Wednesday, June 5
Rhode Island Medical Library

2:00

Call to Order
Welcome by the President
Recognition of Delegates from Maine, New Hampshire, Vermont, Massachusetts and Connecticut.
Report of the Trustees of the Fiske Fund
Report of the Committee on Necrology
Papers:

1. Some Aspects of Convulsive Disorders
   CHARLES P. FITZPATRICK
   Superintendent, State Hospital for Mental Diseases, Howard
2. Observations on Hand Surgery
   TORR WAGNER HARMER
   Instructor in Anatomy, Harvard Medical School
   Assistant Surgeon, Massachusetts General Hospital
3. Adrenal Insufficiency and its Present Day Management
   ROBERT F. LOEB
   Professor of Medicine, Columbia University
4. The Incidence of Diabetes in Rhode Island and Other States
   ELLIOTT P. JOSLIN
   Director of the George F. Baker Clinic, New England Deaconess Hospital, Boston

Evening Session
Rhode Island Medical Library

8:00

Progress in Cancer Control
CLARENCE C. LITTLE, Sc.D.
Managing Director, American Society for the Control of Cancer
Open discussion of the subject in its various phases.

Afternoon Session
Thursday, June 6
Rhode Island Medical Library

2:00

Papers:

1. Leutic Heart Disease in Rhode Island
   CLIFTON B. LEECH
2. Medical Service and the National Health Program
   WALTER G. PHIPPEN
   President of the Massachusetts Medical Society
3. Reduction of Fractures of the Acetabulum with Penetration of the Head of the Femur into the Pelvis—Report of Three cases
   ROBERT L. MAYNARD
   Assistant Professor of Surgery, University of Vermont College of Medicine, Burlington
4. Presidential Address
   CHARLES H. HOLT
5. Induction of Officers for the ensuing year.

Evening Session

6:30

The Annual Dinner,
At the Pomham Club, East Providence, Rhode Island
Anniversary Chairman, JOHN F. KENNEY
Speaker:
REV. ROBERTS A. SEILHAMER
Rector of St. Paul's Church, Pawtucket, R. I.
World Traveller and Lecturer
"The World's Most Northerly Capitol"

LIST OF COMMERCIAL EXHIBITORS AT THE 1940 ANNUAL MEETING of the RHODE ISLAND MEDICAL SOCIETY

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Space 1-16 ..... Main Floor
17-22 ..... Auditorium
23 ..... Dining Hall

RHODE ISLAND MEDICAL SOCIETY

Members of Constituent District Societies

List corrected to April 1, 1940

PAWTUCKET MEDICAL ASSOCIATION

Meets at the Memorial Hospital at 9 P. M. on the third Thursday of each month, September to May, inclusive. Annual Meeting, March, 1940.

President	**Thad A. Krolicki**
Vice President	**G. Raymond Fox**
Secretary	**John H. Gordon**
Treasurer	**Armando A. Bertini**
Councilor	**James L. Wheaton**
Delegates	**Robert T. Henry** **Earl F. Kelly** **Henry J. Hanley** **J. Lincoln Turner**

Fellows

Members of State Society indicated by surname in capitals

BARNES, Albert E.
Beaudoin, L. I.
BENJAMIN, Emanuel W. (Associate)
BERTINI, Armando A.
Burns, Frederic J. (Associate)
CHAPIAN, M. A. (Associate)
CLARKE, Elliott M.
CORMIER, Evariste A.
Doll, Joseph H
DOUCET, Charles S.
DUFRESNE, Walter J.
DURKIN, Patrick A.
FARRELL, Charles L.
Farrell, Irving A.
Fenwick, Adolph R. V.
Ford, Carlton S. (Associate)
Foster, Edward
FOX, G. Raymond
GAUDET, Albert J.
GAYLORD, William A.
GORDON, John H.
Graham, William J. (Associate)
GREENSTEIN, Jacob E. (Associate)
HACKING, Raymond F. (Associate)
Hagan, Eugene A.
HANLEY, Francis E.
HANLEY, Henry J.
Healey, James P.
Healey, Joseph E.
HECKER, Harry
HENRY, Robert T.
Hess, Peter W.
HOLT, Charles H.
HUGHES, Stephen F.
KECHIJIAN, Harry M.
KECHIJIAN, Natalie M.
KELLY, Earl F.
KENNEY, John F.
KENNEY, Stephen A.
KENT, Joseph C.
KROLICKI, Thad A.
LAMOUREUX, S. A.
LAURELLI, Edmund C.
Lussier, Raphael A.
LUTZ, Frank L.
LYNCH, John P.
MARA, Earl J.
MARKS, Herman B.
Marks, Joseph
Marks, Morris
MARSHALL, Julian B.
Masse, Omer H.
MATHEWSON, Earl J.
McCaughey, Edward H.
McClellan, George B.
McGinn, James F.
McGRAW, George B.
McKendall, B. S.
McVay, Frank V. (Honorary)
Mellucci, A. F.
MERDINYAN, Ardashes H.
O'Brien, John H.
O'Brien, Thomas J.
O'Neill, Joseph B.
Paydos, M. W.
PLATT, Marden G.
Robinson, N. D.
Ronne, George E.
Savoie, Joseph U.
SENSEMAN, L. A.
SHERIDAN, James
SMITH, Orland F.
SPRAGUE, Stanley
Stevens, R. E.
Sullivan, James F.
SWEET, Charles F. (Honorary)
Thompson, Edward R.
TOWLE, Bernard L.
TRAINOR, Edward H.
TRIEDMAN, Harry
TURNER, J. Lincoln
Umstead, H. W.
VANCE, Michael E. (Associate)
Vandale, Albert L.
WHEATON, James L.

PROVIDENCE MEDICAL ASSOCIATION

Providence District Society

President.....JOHN G. WALSH
Vice President.....MURRAY S. DANFORTH
Secretary.....HERMAN A. LAWSON
Treasurer.....WILLIAM P. DAVIS

Meets on the first Monday of each month, October to June, inclusive, at the Rhode Island Medical Library

Fellows

Membership in the State Society indicated by surname in capitals

ABBATE, Rocco
ABBOTT, Harlan P.
Abramson, Lewis
ADAMS, Frank M.
Addonizio, Ercole A.
ADELMAN, Maurice
ALEXANDER, George H.
ALLEN, Reginald A.
Allen, Richard E.
ALLIN, Francis E.
Angelone, Carmine T.
ANGELONI, Tito
APPLETON, Paul
Archetto, Angelo
Arciero, Michael
Arlen, Richard S.
ARMINGTON, Herbert H.
ASHWORTH, Charles J.
ASTLE, Christopher J.

Babington, Vernon E.
BALDRIDGE, Robert R.
BARNES, Alvah H.
Baronian, D. Richard
BARR, Kathleen M.
BARROWS, Albert A.
Bartley, James H.
BATCHELDER, Philip
BATES, Reuben C.
BEARDSLEY, J. Murray
Beck, Irving A.
BECKETT, Francis H.
Bell, D. William J.
Bellano, George W.
Bellino, Antonio
BELLIOTTI, Joseph L.
BENJAMIN, Emanuel W.
BERNARDO, John R.
Bernasconi, E. Joseph
BERNSTEIN, Perry
BERRILLO, Anacleto
BEST, Oliver F.
Bianchini, Vincent A.
BIRD, Clarence E.
BISHOP, E. Wade
BLACK, Edward J.
BLANCHARD, Howard E.
BLOUNT, Samuel G.
BOLOTOW, Nathan A.
BOLSTER, John A.
BOURN, Lucy E.
BOWEN, Earl A.
BOYD, James F.
BRACKETT, Edward S.
BRADLEY, Charles
BRADSHAW, Arthur B.
BRAY, Russell S.
Brennen, Earle H.
Breslin, Kate E.
Breslin, R. H.
BROADMAN, Harry
BROTHERS, John H.
Brown, A. A.
BROWN, Frederick N.
Bruno, C. Paul
BUFFUM, William P.
BUGBEE, Raymond G.
BURGESS, Alexander M.
BURKE, Edward F.
BURNS. Francis L.
Burns, Frederic J.
BURNS, Louis E. (Associate)
Burrows, Ernest A.
BURTON, Kenneth G.
Butler, William J.
BUXTON, Bertram H.

Caldarone, A. A.
CALDER, Harold G.
CALISE, Domenico
CAMERON, Edward S.
CAMPBELL, Edward
Capobianco, Giovanni
CAPWELL, Remington P.
CASE, Jarvis D.
CASTALLO, Salvatore
Castronovo, Joseph
Catullo, Emilio A.
CHAFEE, Francis H.
Chapas, Benedict
CHAPIAN, Mihran A.
CHAPIN, Charles V.
CHASE, Peter P.
CHESEBRO, Edmund D.
Cianci, Vincent A.
CICMA, Haralambie G.
Clark, Robert A.
CLARK, Samuel D.
CLARKE, B. Earl
CLUNE, James P.
Cohen, Leo
COHEN, William B.
Coleman, George V.
Collins, Charles M.
Conde, George F.
Connor, Hilary J.
CONRAD, E. Victor
Conte, Alfred C.
CONWAY, John J.
COOK, Irving S.
COOK, Paul C.
COOKE, Charles O.
COONEY, John P.
Corcione, Mary B.
CORRIGAN, Francis V.
CORSELLO, Joseph N.
CORVESE, Anthony
COUGHLIN, Fred A.
COX, James H.
Crane, G. Edward
CRANK, Rawser P.
CUMMINGS, Frank A.
CUMMINGS, William W.
CURREN, L. Addison
Curreri, Gerald J.
CUTTS, Frank B.
CUTTS, Morgan
CUTTS, William B.

Damarjian, Edward
DANFORTH, Murray S.
D'Angelo, Antonio F.
Davis, George W.
DAVIS, William P.
DeCesare, Francis A.

DEERY, James P.
DeFusco, Bruno G.
Del Selva, Americo
DeNyse, Donald L.
DEVERE, Frederick H.
DE WOLF, Halsey
DI LEONE, Ralph L.
DIMMITT, Frank W.
DiPippo, Palmino
Dolan, Thomas J.
DONLEY, John E.
Donnelly, John J.
DOTEN, Carl R.
DOWLING, Joseph L.
D'Ugo, William P.
DUSTIN, Cecil C.
DWYER, George J.
Dziob, John S.

Earley, Charles P.
ECKSTEIN, Adolph W.
EDDY, Jesse P., 3rd
Egan, Thomas A.
Eliot, Alice M. B.

FAGAN, James H.
Fain, William
Fallon, James T.
FARRELL, John T.
FARRELL, Robert L.
Feifer, Anthony M.
FEINBERG, Banice
Femino, Richard
FERGUSON, John B.
Ferrara, Bernardino F.
FIDANZA, Antonio G.
Field, Eugene A.
FISHBEIN, Jay N.
FITZPATRICK, Charles P.
FLETCHER, William
FLYNN, Harry S.
FLYNN, Joseph C.
Foley, William H.
FORGET, Ulysse
Fortunato, Stephen J.
FOX, A. Henry
FOX, G. Raymond
Fracasse, John
FRANKLIN, Joseph
Fratantuono, Frank D.
FREEDMAN, David
Freedman, Stanley S.
Fuhrmann, Louis J.
FULTON, Frank T.

GALLAGHER, Henry J.
Gannon, C. H.
GARSIDE, Francis V.
GEIGER, Preston D.
GERBER, Isaac
Giannini, Pio
GIBSON, J. Merrill
GIFFORD, Nathaniel H.
GILBERT, James A.
GILBERT, John J.
Gillis, Nora P.
Giura, Arcadie
GOLDBERGER, Milton
GOLDOWSKY, Seebert J.
Golini, Carlotta N.
Goodman, Louis
GORDON, Walter C.
GORMLY, Charles F.
Gormly, John A.
Granger, Eugene N.
GREENSTEIN, Jacob
GREGORY, Kalei K.
Gross, C. R.
GROSSMAN, Herman P
GROVER, Morris L.
GRZEBIEN, Thomas W.
Gulesserian, Hampartzum S.

HACKING, Raymond F.
HALE, Frank S.
HALL, Hugh J.
HAM, John C.
HAMILTON, James
HAMMOND, Roland
HANSON, F. Charles
HAPP, Linley C.
HARDMAN, Margaret S.
HARDY, Arthur E.
Harrington, Harold F.
Harrington, Peter F.
HARRIS, Herbert E.
HARVEY, N. Darrell
HASCALL, Theodore C.
Haverly, Richard E.
HAWKES, Charles E.
HAWKINS, Joseph F.
HAYES, Walter E.
HAYWARD, John A.
Heffernan, Edward V.
HILL, Prescott T.
HINDLE, William
HODGSON, William H.
Hoey, John J.
Hoey, Waldo O.
HONAN, Frank J.
HOPKINS, Henry W.
HORAN, William A.
HOUGHTON, Montafix W.
HOUSTON, Craig S.
HOYE, Henry J.
Hubbard, John D.
HUGHES, William N.
HUNT, Russell R.
HUSSEY, Frederic V.
HYER, Harrison F.

Iavazzo, Anthony A.
Indeglia, Pasquale V.

JACKVONY, Albert H.
JACOBSON, Frank J.
JOHNSTON, Joseph C.
JONES, Henry A.
JONES, Walter S.
JORDAN, Harmon P. B.
JORDAN, William H.
JOYCE, Henry S.
Juracsek, Valeria R.

KECHIJIAN, Harry M.
Keefe, Howard F.
KEEFE, Patrick H.
KELLEY, Jacob S.
KENNEY, John F. (Associate)
KENNEY, John J.
Kennison, Samuel I.
KENNON, Charles E. V.
Keohane, John T.
KIENE, Hugh E.
KINGMAN, Lucius C.
KRAEMER, Richard J.
KRAMER, Louis I.

LANGDON, John
Lange, H. A.
LAURELLI, Edmund C. (Associate)
LAWSON, Herman A.
Lawton, Anne L.
LEECH, Clifton B.
LEET, William L.
LENZNER, Simon G.
Lewis, Charles B.
LIBBY, Harold
Lippitt, Louis D.
Lisbon, Wallace
Litchman, David
LITTLEFIELD, Frank B.
Londergan, John F.
Longfellow, A. H.
LORD, Robert M.
LUONGO, Fedele U.

McCABE, Francis J.
McCAFFREY, Jerome J.
McCANN, James A.
MacCARDELL, Frank C.
McCOART, Richard F.
McCURDY, Gordon J.
McCUSKER, Henry
McDONALD, Charles A.
McEVOY, Frank E.
McGUIRK, William R.
McIsaac, John C.
McKendall, B. S. (Associate)

McKendry, James R.
McLAUGHLIN, Edward A.
MAGILL, Wm. H.
MAHONEY, Andrew W.
MAHONEY, William A.
Mankis, George R.
MARGOSSIAN, Arshag D.
Marsh, John H.
MARTIN, Arthur E.
MARTIN, Thomas A.
MARTINEAU, Lawrence A.
Marzilli, Alexander F.
MATHEWS, Frank H.
MATHEWS, George S.
MATHEWSON, Earl J. (Associate)
MATTEO, Frank I.
Mattera, Vincent J.
Mellone, John A.
MELVIN, Edward G.
Menzies, Gordon E.
Menzies, John E.
MERCHANT, Marcius H.
Merlino, Frank A.
MESSINGER, Harry C.
Miga, Casimir J.
MIGLIACCIO, Anthony V.
MILAN, Michael B.
MILLER, Albert H.
Miller, Himon
MILLS, Parker
MINER, Harold C.
MISSIRLIAN, Mihran
Molony, Walter J.
MONAHAN, John T.
Monti, Emilio J.
MOOR, Henry B.
MOORE, James S.
Moore, Kenneth T.
MOREIN, Samuel
MORI, Laurence A.
MOWRY, Classen
MOWRY, Jesse E.
Mulvey, William A.
MUNCY, William M.
MURPHY, John F.
MURPHY, Robert G.
Murphy, Thomas H. (Providence)
MURPHY, Thomas H. (Pawtucket)
Myrick, John C.

NESTOR, Michael J.
NEWSAM, Arthur R.
NICHOLS, Ira C.
Normandin, Louis A.
NOURIE, Joseph P.
NOYES, Ira H.

O'Brien, John H.
O'CONNELL, Francis D.
O'CONNELL, Joseph C.
O'Connell, Thomas L.
O'CONNOR, Michael J.
ODDO, Vincent J.
O'Donnell, Alan E.
O'Reilly, Edwin B.
O'Meara, Catherine E.
O'ROURKE, Charles B.
O'ROURKE, Patrick I.

PALMER, William H.
PARKINSON, James McD.
PARTRIDGE, Herbert G.
PEARSON, Rudolph W.
PEDORELLA, Americo J.
PELLETIER, Emery
Penington, Robert, Jr.
PERKINS, Jay
PETERS, John M.
PETRUCCI, Ralph J.
PHILLIPS, Charles L.
PICKLES, Wilfred
Picozzi, John A.
Pinckney, John I.
PITTS, Herman C.
PORTER, Emery M.
PORTER, Lewis B.
Portnoy, Bradford M. S.
POTTER, Alfred L.
Potter, Charles
POTTER, Merle M.
Potter, Walter H.
Pournaras, Nicholas A.
Pozzi, Gustave
PRIOR, James H.
Pritzker, Samuel

QUESNEL, Ernest J.

Raia, J. E.
Rakatansky, Nathan S.
Rattenni, Arthur
RAYMOND, Charles N.
REGAN, John F.
REGO, Rodrigo P. da C.
REGO, Victor P. C.
Ricci, Edward A.
RICE, William O.
RICHARDSON, Dennett L.
RILEY, Clarence J.
Riley, Frederick R.
RITTNER, Mark
Roberts, William H.
Robinson, Nathaniel D. (Associate)
ROBINSON, Robert C.
ROGELL, Harold
Romano, Anthony
RONCHESE, Francesco
ROSE, Alanson D.
ROSS, Florence M.
Ross, Margaret B.
Rossignoli, Vincent P.
ROUNDS, Albert W.
Rozzero, Paul J.
Ruest, Florian G.
RUGGLES, Arthur H.
Ruhmann, Edward F.
Russell, Amy E.
Ryan, Jerome J.
RYAN, Vincent J.

Sage, Louis A.
Saklad, Elihu
SAKLAD, Meyer
SAKLAD, Sarah M.
Sammartino, Agostino
SANBORN, Harvey B.
Sannella, Lee G.
Sarafian, John C.
SARGENT, Francis B.
SAVRAN, Jack
SAWYER, Carl D.
SAYER, Edmund A.
SCANLAN, Thomas F.
Schradieck, Constant E.
Schwab, William J.
SCORPIO, Angelo
Scotti, Ciro O.
Seltzer, Edward I.
SHARP, Benjamin S.
SHARP, Ezra A.
SHATTUCK, George L.
SHAW, Eliot A.
Shea, Richard L.
Sheehan, John J., Jr.
Sheridan, Thomas P.
Sherman, Bernard I.
SHIELDS, William P.
SIELKE, Eugene L.
SMITH, Clara L.
SMITH, Joseph
SMITH, Orland F. (Associate)
SMITH, Thomas J.
SOUTHEY, Charles L.
SPERBER, Perry
Starr, Samuel
STEPHENS, Henry F.
Stone, Edgar F.
STONE, Ellen A.
STONE, Eric P.
STREKER, Edward T.
Streker, John F.
STREKER, William S.
STURGIS, Karl B.
Sullivan, Ralph V.
SWEENEY, John W.
SWEET, Charles F.
Sydlowski, Edmund J.
SYLVIA, Charles A.

TAGGART, Fenwick G.
Tarro, Michael A.
Temple, Francis E.
THOMPSON, Edwin G.
Thompson, Ernest D.
TINGLEY, Louisa P.
Topaz, Anna
TROPPOLI, Daniel V.
Trottier, Arthur O.
TURNER, Charles S.
TURNER, Howard K.

UTTER, Henry E.

Vallone, John
VAN BENSCHOTEN, George W.
Vaughn, Arthur H.
Vieria, Edwin

WALSH, John G.
Warren, Jacob P.
WATERMAN, George W.
WEBBER, Joseph B.
WEBSTER, Frederick A. (Associate)
WEIGNER, Walter C.
WELCH, Stephen A.
WELLS, Guy W.
West, Edward J.
WESTCOTT, Clinton S.
WESTCOTT, Niles
WEYLER, Henry L. C.
White, Charles E.
WHITE, George F.
WHITMARSH, Robert H.
WILCOX, Roswell S.
WILLIAMS, Harold W.
WILLIAMS, P.
Williams, Robert J.
WINDSBERG, Eske H.
WING, Elihu S.
WINKLER, Herman A.
WINKLER, Malcolm
WISE, Bernard O.
WOODMANSEE, Clarence H.

Yessian, Mark A.
Young, Daniel D.
YOUNG, George L. (Associate)

ZAMBARANO, Ubaldo E.
Zinno, Genarino R.
Zouraboff, Catherine
Zurawski, Charles

KENT COUNTY MEDICAL SOCIETY

Annual Meeting — 2nd Thursday in December

President	**Stanley D. Davies**
Vice President	**Joseph E. Wittig**
Secretary	**Whitman Merrill**
Treasurer	**J. Fulgence Archambault**
Councilor	**Rocco Abbate**
Delegate	**Rocco Abbate**

Fellows

Membership in the State Society indicated by surname in capitals

ABBATE, Rocco
ARCHAMBAULT, J. Fulgence
Baute, Joseph A.
CHRISTIE, Charles S.
COLLOM, Harold L.
DAVIES, Stanley D.
Duquette, Leo H.
Dyer, William H.
ERINAKES, Peter C. H.
Farrell, George B.
HARDY, Arthur E.
Hemond, F. J.
HUDSON, Royal C.
Kostyla, Edward A.
LUFT, Raymond
LUPOLI, Alphonse W.
MACK, John A.
MERRILL, Whitman
PHILLIPS, Charles L.
Senerchia, G.
SMITH, R. Morton
Spearman, L. L.
TAGGART, Fenwick G.
TEFFT, Benjamin F.
Wittig, Joseph E.
YOUNG, George L.

NEWPORT COUNTY MEDICAL SOCIETY

Annual Meeting held February 1, 1940

President	**Samuel Adelson**
First Vice President	**Philip Geller**
Second Vice President	**Louis E. Burns**
Secretary	**Alfred M. Tartaglino**
Treasurer	**Norbert U. Zielinski**
Councilor	**Charles W. Stewart**
Delegates	**Samuel Adelson, Louis E. Burns**

Fellows

Membership in the State Society indicated by surname in capitals

ADELSON, Samuel
Arlen, Richard S.
BURNS, Louis E.
Butler, Maurice
CALLAHAN, James C.
Ciarla, P. P.
Clarke, Philip E.
CORBETT, Francis A.
Creamer, William H.
De Blois, Seth
DOTTERER, Charles S.
GELLER, Philip S.
Healy, John L.
JACOBY, Douglas P. A.
King, Arthur
Lent, J. W.
MacLEOD, Norman M.
McCarthy, Eugene
Ramos, Jose
Redman, William L.
Smith, Daniel A.
STEWART, Charles W.
Stoops, William A.
SULLIVAN, Michael H.
TARTAGLINO, Alfred M.
Tennis, Matthew N.
Walsh, James
YOUNG, John A.
Zielinski, Norbert U.

WASHINGTON COUNTY MEDICAL SOCIETY

Annual Meeting — January, 1941

President	**Michael H. Scanlon**
First Vice President	**Linwood H. Johnson**
Second Vice President	**A. L. Manganaro**
Secretary and Treasurer	**Julianna R. Tatum**
Councilor	**John Paul Jones**
Delegates	**John W. Helfrich, Freeman B. Agnelli**

Fellows

Membership in the State Society indicated by surname in capitals

AGNELLI, Freeman B.
BARBER, Joseph D.
BURKE, Francis E.
CERRITO, Louis C.
Crandall, Charles P.
CRANDALL, Harry F.
Davis, Paul V.
Depner, Rudolph J.
DUCKWORTH, Milton
FARAGO, Samuel S.
FITTS, Fernald C.
Gammell, Edwin B.
GONGAWARE, Hartford P.
GRENOLDS, Walter J.
HATHAWAY, Clifford S.
HELFRICH, John W.
JOHNSON, Linwood H.
JONES, John Paul
Kenyon, Frances A.
KENYON, Harold D.
LADD, Joseph H.
LASKEY, Howard G.
Manganaro, A. L.
Manning, P. J.
Mastrobuono, A.
McAteer, R. F.
Menzies, Gordon
Morrone, Louis
NATHANS, Samuel
Potter, Henry B.
RUISI, John E.
SCANLON, Michael H.
TATUM, Julianna
THEWLIS, Malford W.
Thompson, William C.
TURCO, Salvatore P.
VISGILIO, Thomas, Jr.
Webster, Samuel C.

WOONSOCKET DISTRICT MEDICAL SOCIETY

Annual Meeting held March 26, 1940

President	**Victor H. Monti**
Vice President	**Guyon G. Dupre**
Secretary	**Thomas J. Lalor**
Treasurer	**Joseph W. Reilly**
Councilor	**John V. O'Connor**
Delegate	**Leo V. Conlon**

Fellows

Membership in the State Society indicated by surname in capitals

ASHTON, George W.
Barry, Cornelius B.
Bertone, Virgilio M.
BOUCHER, Paul E.
CHARON, Ernest A.
Cicchetti, John R.
CLARKE, Elisha D.
CONLON, Leo V.
CONSTANTINEAU, Aurelien
Crepeau, George
DOWLING, Richard H.
DUGAS, Leo
DUPRE, Guyon G.
EDDY, Augustine W.
EMIDY, Herman L.
EMIDY, Stephen E.
FLYNN, Thomas S.
FONTAINE, Auray
GARRISON, Norman S.
GAUTHIER, Henri E.
ISRAEL, Cyril
Kaskiw, Emil A.
KENNEDY, Thomas F.
King, Alfred E.
KING, Francis J.
KING, William A.
LALOR, Thomas J.
McCARTHY, James M.
McCOOEY, James H.
McKenna, Joseph B.
MEDOFF, Edward B.
MONTI, Victor H.
O'BRIEN, James P.
O'CONNOR, John V.
PARDEE, Katherine
POTTER, Edgar S.
REILLY, Joseph W.
ROCHELEAU, Walter C.
ROSWELL, Joseph T.
TANGUAY, Joseph E.
WEEDEN, Allen A.
WITTES, Saul A.